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THE TREATMENT OF NEUROSYPHILIS.[1]

By ERIC SUSMAN,
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IN introducing the subject of the therapeutics of neurosyphilis I should like you to note that there is a man, living and working with us here in Sydney, who is a significant link with the past. That Nestor of Australian medicine, Robert Scot Skirving—and, incidentally, sometime house surgeon to Argyll-Robertson—was wont to treat locomotor ataxia with pills of silver nitrate. In his time he has prescribed these pills by the bucketful. So I want to make the point that, actually within living memory, we have travelled far along the therapeutic road. But the journey has not been made at a steady, even pace. Rather has it been made by fits and starts, in a very jerky, spasmodic fashion.

Now I want to borrow an expression, recently revived and popularized by a well-known socialist politician. For syphilologists the first decade of this century was truly a Golden Age. Almost within a matter of months the brilliant Schaudinn discovered the *Treponema pallidum*, the ingenious Wassermann applied complement fixation methods to work out his diagnostic test, and the painstaking Ehrlich, after hundreds of experiments, produced a powerful antisyphilitic remedy. What an exciting period it must have been! The causative organism, a reliable laboratory test and a specific cure, all in one fell swoop! This ancient dreaded scourge, "half as old as Time", was right "in the bag"!

Somehow things did not work out quite according to plan. Patients were given two or three injections of "606" and, hey presto! the chancres and the cutaneous syphilides disappeared in an unbelievably magical, necromantic way. The delighted customers were sent about their business and went on their ways, rejoicing. Aortic aneurysm, aortic regurgitation, *tabes dorsalis* and optic atrophy were still clinical commonplaces in the wards of general hospitals, and the lunatic asylums continued to house a goodly proportion of general paralytic boarders. Disappointingly, the Golden Age had taken on a silvery or bronze hue. The next important milestone was the introduction of malaria in the treatment of *dementia paralytica* by Wagner-Jauregg.

Another kaleidoscopic era, similar to those just mentioned, started in 1943, when Mahoney and his co-workers introduced penicillin, with brilliant results, in the treatment of early syphilis. When supplies of the drug became more plentiful, many workers the world over began to use it in the treatment of various forms of neurosyphilis, and it soon became apparent that we had in our hands an amazingly efficacious remedy. It is our task, in our discussion this evening, to take stock of the present position. In discussing the treatment of any disease it is, I think, permissible to say a word on its diagnosis. When syphilis involves the nervous system this involvement may be minimal and inconspicuous. This is latent neurosyphilis. As in other disseminated types of disease, all depends on the lottery or chance of the site of the lesion. Some lesions exteriorize their presence very readily; others remain silent. In the second place, neurosyphilis may present itself as a more or less easily recognized pattern, such as general paralysis of the insane or *tabes dorsalis*. Thirdly, there is the syndrome of meningo-vascular syphilis, with its rich, changeable, fluctuating symptom-

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 29, 1949.

atology. Even before this specialized audience I must stress the fact that there is only one answer to correct diagnosis: an examination of the cerebro-spinal fluid. I have made and have seen made many mistakes by the omission of this quintessential laboratory aid. Here is an illustration.

CASE I.—A previously healthy man, aged fifty-two years, of dissolute habits, and a great lover of tavern life, one night drank more than his usual quota of alcohol. He did not remember very much about going home, but it was evidently a stormy passage, because he did recollect falling over twice. He awakened in bed next morning, fully clothed, and having wet himself. His head ached severely, which was understandable, and he was very thirsty, which again was not surprising. It soon became apparent to this experienced toper that this was no ordinary "hang-over", because these two symptoms, headache and thirst, persisted for many days and became worse. Within a week, his fluid intake was measured in gallons, he lost weight, he could get no rest because of thirst and frequent urination. He rapidly became a pituitrin addict. His medical attendant diagnosed *diabetes insipidus* of traumatic origin, postulating a lesion in the pituitary region, due to a small basal fracture or a meningeal tear sustained in one of his tumbles on the evening of the onset of his illness. We were obliged to revise the diagnosis when the cerebro-spinal fluid was found to be characteristically syphilitic. The patient made a brilliant and rapid recovery after treatment by penicillin.

Here is another instructive experience:

CASE II.—A respectable widow, aged fifty years, without rhyme or reason began to wet the bed. Cystoscopy showed that the bladder was of less than average capacity, and some form of bladder reeducation was prescribed. Her bed wetting did not diminish. Warming up to his work, the urologist cauterized one or two suspicious-looking spots on the bladder wall, the nature of the lesions not being very clear. Her distressing and humiliating symptom persisted. Eventually the patient consulted a doctor. I was unable to fault her nervous system. The diagnosis became obvious when cerebro-spinal fluid studies revealed her to be a syphilitic. She responded poorly to penicillin treatment, and there was still further confirmation of the diagnosis, if such were necessary, in the slow development of optic atrophy.

It may be useful at this point to comment on the significance of the abnormal findings in the cerebro-spinal fluid. The positive Wassermann reaction means that the patient has, or has had, neurosyphilis. An increased cell count means that active inflammation is present. Any count above five cells per millilitre is definitely abnormal. A high total protein content also speaks for activity, and a failure of the level to fall suggests that treatment has not been adequate. The Lange curve attempts to decide whether the disease is mostly parenchymal, or mainly inflammatory. It is not, in my view, of the importance of the other three tests.

Occasionally neurosyphilis is diagnosed when in fact it is not present. No discussion of the treatment of neurosyphilis would be complete without mention of the false positive Wassermann reaction. I hope this important subject will be brought up by speakers competent to do so, and I simply draw your attentilon to the subject by quoting the following case.

CASE III.—A man, aged thirty-two years, was admitted to hospital in *status epilepticus*. For the past nine months he had been having seizures at the rate of about one a month. They were mostly nocturnal, of the *grand mal* type, and there was no aura. A maternal grandmother and a sister suffered from epilepsy. The patient had one healthy child, and his wife had had no miscarriages. On one occasion, the Wassermann test had produced a positive reaction in the cerebro-spinal fluid. After his first seizure, he was treated for cerebral syphilis with arsenic and bismuth, but the seizures continued at fairly regular intervals. For his first six days in hospital the patient averaged six major attacks *per diem*, and then they gradually became fewer. There were no abnormal physical signs, other than the seizures, and the cerebro-spinal fluid was normal in all respects. The electroencephalographic findings were consistent with the diagnosis of idiopathic epilepsy, and the patient responded admirably to the exhibition of "Dilantin" and phenobarbital.

Treatment.

The prophylaxis of neurosyphilis is simple to state, but difficult to achieve. It is the early and thorough treatment of the primary disease. In a big seaport terminal, such as Sydney, there must be many hidden reservoirs of infection.

Some years ago, by shrewd detective methods, a colleague found that nine successive cases of chancres had arisen from a common source, in the person of a "teen-age blonde" who worked as a cloakroom attendant at one of the lesser-known night clubs. It was a serious matter, because this girl had the reputation of conducting a stupendous practice. It was regarded as a little more than coincidental that a visiting American squadron had recently gone to sea.

I do not know if such conditions obtain at the present time.

American workers appear to be worried about the problem of reinfection. It must be difficult to distinguish between reinfection and reactivation, and they speak of "ping-pong" (or, better, "see-saw") syphilis—meaning that, by alternation, sporting consorts reinfect each other. Just so long as the laity take a flippant, airy view of venereal disease will syphilis be with us and, therefore, neurosyphilis. When the abnormal fluid changes characteristic of secondary syphilis persist it means that such patients are candidates for subsequent neural involvement. If at the end of one year's treatment the blood and fluid are normal, then neurosyphilis is most unlikely to arise.

And now, after this long pedestrian preamble, we come to the actual treatment. It is well nigh impossible to assess correctly the present position of penicillin in the treatment of neurosyphilis. There are so many variables. Dosage and dosage spacing are not standardized, new (and presumably more efficacious) types of penicillin with prolonged absorption time are being introduced almost every month, and most reports are contradictory and lack statistical value. It may be useful to telescope the present therapeutic position. All are agreed on the following points:

1. Penicillin can do in days and weeks what arsenic and bismuth could do in months and years.
2. Penicillin acts most efficaciously in that type of neurosyphilis which was so responsive to the older treatments—namely, meningo-vascular syphilis.
3. No drug or treatment, penicillin included, can resurrect dead mechanisms. Nothing can exhume a neuron that has perished. In other words, the greater the parenchymatous involvement, the poorer the therapeutic response.
4. Because of its tendency to "burn out", the treatment of *tabes dorsalis* is notoriously difficult to assess.
5. Intrathecal therapy is unnecessary.

This is the story of a 1947 patient who was hopelessly overtreated.

CASE IV.—A bartender, aged thirty-five years, had suffered for five years from dribbling of urine and precipitate micturition. He seemed to have put up with these symptoms until a few months before his admission to hospital, when he suddenly lost the use of his legs, and experienced lightning pains, gastric crises and various parasthesiæ. On examination, he showed all the signs of advanced *tabes dorsalis*. On account of his comparative youth and the rapidity of the disease process, it was decided to treat him with great vigour. He was given 1,000,000 units of penicillin intramuscularly daily, and 100,000 units intrathecally, for six days. At the end of this ordeal his condition was pitiable. He had a high fever, rapid pulse and neck stiffness, and vomited incessantly. There were 2000 cells per millilitre in his cerebro-spinal fluid, and we despaired of his life. However, after a long and tedious convalescence, he recovered, and once he was on his feet, six weeks after his treatment commenced, it was observed that his ataxia had decreased in a really remarkable manner, and that Argyll-Robertson pupils had become normal—a happening unique in my experience.

It is the management of *dementia paralytica* that is in dispute and it all comes down to the question of fever *plus* penicillin *versus* penicillin alone. Here are two extracts from the current literature, illustrating the widely divergent views that are held on the subject. Nicol, who has worked at the Malaria Therapy Centre in England for many years, writes as follows of penicillin treatment in general paresis:

> One of the most striking clinical features was the physical and mental improvement in many patients, a

phenomenom rarely, if ever, seen during malaria therapy. Most dramatic results were seen in patients who were confused, and in poor physical condition.

Contrast this experienced English opinion with an authoritative American view by O'Leary:

I have never seen a clear-cut remission develop in a patient with frank dementia paralytica of any type from penicillin alone, and the partial remissions that occur not only are incomplete, but are of short duration. However, penicillin plus fever therapy produce complete remission.

Mr. President, you may take your pick.

Spinal Syphilis.

Spinal syphilis is a rare and frightful variant of neurosyphilis. In the last three years I have seen only six cases. Some patients were treated conservatively and timidly, others more boldly. In spite of treatment all six patients became spinal animals under our very noses and all died. It would be interesting to know if anyone has treated with anticoagulants these people suffering from syphilitic meningo-myelitis with cord thrombosis.

Summary.

For purposes of discussion I will now summarize my own views.

1. The place for tryparsamide is the waste-paper basket.

2. Arsenic and bismuth are obsolescent. I use bismuth courses simply to keep patients under observation and as a *placebo* for certain syphilophobics.

3. If penicillin alone is used, an ordinary standard course is 9,000,000 to 12,000,000 units, spread over two weeks.

4. There is no excuse for using penicillin intrathecally for any form of neurosyphilis.

5. It is an error to undertreat neurosyphilis. No patient should be pronounced "Wassermann-fast" until he has had repeated courses over a period of two years.

6. Herxheimer reactions should be ignored. This term does not embrace, of course, penicillin rashes or other evidence of penicillin sensitivity.

7. On the information at present available the treatment of choice is the combination of penicillin and malaria. The fever should be given two or three days' start on the penicillin.

8. It seems reasonable to treat residual psychoses by shock therapy.

Conclusion.

To conclude, Mr. President, from the long story of the disease it would appear that man is ideally suited to the requirements of the spirochæte. What is it in man's chemical make-up that makes him such a tasty, succulent morsel for the parasite? We must await the answer to that question until the organism has been successfully cultivated and until its microchemistry has been intensively studied. We may congratulate ourselves on having made great progress in the treatment of the disease. But please do not imagine that we have the complete answer. In this atomic age one may speculate that Ehrlich's Utopian conception of the *dosis sterilisans magna* will be found in the isotopes.

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THE TREATMENT OF NEUROSYPHILIS.

By E. T. HILLIARD, M.B., Ch.M., D.P.M.,
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IN any outline of the treatment of neurosyphilis it is necessary to preserve an unbiased mind and to detail the methods of treatment and their results, leaving the decision about any individual therapy to the observer. A review of the literature leaves one with the feeling that many observers, in advocating their own methods of treatment, develop a bias towards others.

It was soon found that the treatment of syphilis with arsenic, mercury, bismuth or iodides could not be extended with equal success to neurosyphilis. In the early twenties the infirmary wards of mental hospitals contained numbers of sufferers from neurosyphilis in the final stage of general paralysis, who were faulty, demented and paralysed. Yet such patients had been given full courses of antisyphilitic treatment without success. Patients treated in the early stages had a better outlook, and hospital records placed the recovery rate at 6% to 10%.

It had been noticed for some time that the occurrence of some infection associated with high fever in a sufferer from general paralysis had a beneficial effect and even led to recovery. Erysipelas, pneumonia and typhoid fever had all been noted to cause recovery. Von Jauregg had long toyed with the idea of inoculating general paralytics with erysipelas or malaria, and when in 1918 a soldier, wounded on the Serbian front, developed malaria, his infected blood was injected into a sufferer from neurosyphilis and a new era of treatment was born.

Malarial treatment was introduced to this State in the mid-twenties, and the late Clifford Henry often recounted with pride the first successful inoculation of malarial blood delivered by aeroplane.

Shortly afterwards, the use of tryparsamide, a pentavalent arsenical compound, was introduced, followed by the intravenous use of "T.A.B." vaccine.

The use of these newer methods of treatment increased the recovery rate to a figure varying from 30% to 40%, according to the claims of different observers. In those patients who did not recover, the progress of the disease was arrested or greatly slowed down. The old name of general paralysis fell out of usage and the term "*dementia paralytica*" was substituted.

In the early thirties the accent in treatment fell on methods to produce heat by electrical means and diathermy, and the "Inductotherm" came into use. Figures for this treatment placed the recovery rate at 40%, a further 42% of patients being relieved.

The advent of penicillin for the treatment of coccal infections was followed by its use for attacking the spirochæte, and in some clinics this is at present the only method of treatment.

The various forms of treatment come under two main headings: (*a*) drug therapy, (*b*) physical therapy (fever-producing).

With the failure of "Salvarsan" therapy to produce better effects than the use of mercury and iodides, efforts were made to introduce the drug to the central nervous system directly through the use of "Salvarsanized" serum. This was injected intrathecally in the lumbar region and later into the *cisterna magna* and even into the lateral ventricles. But the trivalent arsenic proved too toxic for general use, and further progress in the use of arsenicals had to wait for the advent of the pentavalent compound tryparsamide. This is injected intravenously in doses of

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two to three grammes dissolved in 10 millilitres of freshly distilled water every week for eight weeks. Three to four courses of tryparsamide may be necessary to effect a cure. Its success may be attributed to its ability to pass the barrier of the chorioid plexus. The resting period between courses of tryparsamide may be filled by intramuscular injections of "Bismol", with the administration of mercury and iodide by mouth.

The drawback to tryparsamide is its action on the optic nerve fibres, and the drug should never be given without a preliminary ophthalmological examination. Even if this reveals no abnormality careful watch should be kept for any constriction of the visual fields or the subjective experience of dazzling vision. This may occur from the third to the sixth injection, and if this period is passed it does not usually occur later.

Recently a new pentavalent arsenical, "Aldarsone", has been introduced. This is injected intravenously in doses of 0·25 gramme dissolved in 10 millilitres of distilled water, the average being increased to 1·0 gramme later, and given weekly for twenty-four weeks. Eisenberg and Hershfield (1948) combine this with one millilitre of bismuth subsalicylate solution given intramuscularly.

It is hoped that the present optimism regarding the efficiency of penicillin does not retard future progress in chemotherapy in neurosyphilis. Some observers have discarded pentavalent arsenicals because of the detrimental effect on the macula or on the optic nerve fibres, but this effect is much questioned and is rejected by Sexton, of Canada. The chronic inflammatory process of optic atrophy begins at the margins, causing concentric narrowing of the visual fields, and it causes degeneration by penetration along the septal tissues and the blood vessels of the optic nerves. Visual field constriction occurred in only one case of the series of 54 cases of neurosyphilis treated with "Aldarsone", and on discontinuance of the drug no further visual impairment was noted.

Malarial Treatment.

Two millilitres of blood are injected intramuscularly between the shoulders. After the seventh day a four-hourly record is kept; disturbances of temperature will occur after the eighth day and in a further two or three days the characteristic intermittently elevated temperature, quotidian in type, establishes itself. The height and duration of the paroxysms must be carefully noted. After ten to twelve days of temperature over 103° F. the fever is arrested by quinine. The temperature should not be allowed to rise over 105° F. and fluids should be given freely. Blood films should be examined from the fourth day onwards.

After the cessation of treatment the patient is frequently exhausted and needs building up with iron, liver extract and vitamins. Recurrences of malaria have to be watched for and treated with quinine, five grains daily for four to seven days.

After treatment (if successful) it is noted that the patient has forgotten many of his old expansive delusions and the confusion clears up. The recovery may not be perfect, as certain cortical damage may have become irreversible through delay in treatment, and a mild permanent dementia results. On the other hand, the removal of the florid psychiatric symptoms of general paresis may release an underlying non-organic psychotic syndrome, such as a manic-depressive psychosis, which may well require treatment by electro-convulsive therapy.

As soon as possible, efforts should be made to return the patient to his former environment, care being taken that regular checking of the cerebro-spinal fluid can be conducted at regular intervals.

Although relapses can occur up to three months after apparent recovery, these are rare if two successive testings of cerebro-spinal fluid at three-monthly intervals give normal findings. The complications of malarial therapy are jaundice, œdema of the ankles, albuminuria, neuritis and dyspnœa. Jaundice is due to heavy parasitæmia and can be relieved by temporarily arresting the fever. Other complications can be met by the use of "Thio-bismol" in doses of 0·2 gramme, which converts a quotidian fever into a true tertian type and renders the treatment safe (Sargent and Slater, 1948; Nicol, 1948).

Electropyrexia.

In 1929 Neymann and Osborne published their paper on "Artificial Fever Produced by High Frequency Currents" (Eisenberg and Hershfield, 1948). They showed that by electrical means the patient's temperature could be raised to any desired degree and maintained at this degree as long as was thought desirable. This was a great advance over any other method of producing heat. At this time Neymann and Osborne used a specially constructed diathermy machine, capable of delivering 6000 milliamperes. Experience showed that lesser currents could be used with greater safety. An open tungsten spark-gap type was substituted with a milliamperage up to 4000. Large electrodes of tinfoil or lead were used. Later Neymann devised an electrode, backed with rubber, impregnated with minute particles of zinc, and incapable of producing burns. These we have found extremely satisfactory, but they are expensive and not of long life.

The next stage in the development of electrically produced heat was the introduction of the "Inductotherm" machine, which seems to have rendered diathermy, as a means of producing hyperpyrexia, a thing of the past. The "Inductotherm" abolishes the use of electrodes and removes the risk of burns. With it the patient becomes the centre of a magnetic field, and no current passes through him. The heat is produced by eddy currents similar to those used in the induction furnace for the production of high-grade steel.

More recently an air-conditioning chamber has been built. In this the air is heated and humidified and forced to circulate through the chamber in which the patient is placed. It is described by Desjardins, Stuhler and Popp in their paper "Fever Therapy for Gonococcal Infections". With the use of this chamber the patient's temperature is raised to 106° F. in from sixty to ninety minutes.

The clinical application of the "Inductotherm" is simpler than that of the diathermy machine. Three turns of insulated cord are placed round the patient's trunk, after the patient's body has been rolled in blankets or a sleeping bag. We find that the "Inductotherm" generates heat more rapidly and better than either the ordinary diathermy machine or any of the so-called short-wave appliances of which we have knowledge. We attribute the speedier and better controlled heat in the patient to the greater area of his body which is enclosed within the active field. The respiration and pulse charts have been of smaller range since the "Inductotherm" was used.

The technique consists of insulating the patient against the loss of heat. This is done by wrapping him first in a terry cloth to absorb moisture and then in a mackintosh sheet, after which he is folded in from seven to nine blankets, only the face and head being left exposed to the air. The current is turned on until the patient's temperature is about 103° F. This will take from two to four hours. After the current is turned off the temperature will continue to rise another two, three or more degrees, and in a favourable case it will be maintained until the patient is uncovered. If the temperature falls too early it can be again raised by turning on the current. Individuals react very differently, both as to the rate of rise of the temperature and as to the height it reaches after the current has been turned off. On rare occasions the unexpected happens and a temperature of 107° F. will be recorded. On some occasions the symptoms have been alarming, but never fatal. The fever is easily controlled by removing the covering and sponging the patient.

The following is our routine method.

The treatment bed is arranged in the following manner. Two half mattresses are placed on top of the regular mattress (avoid the use of wire—a wooden bed spring is essential). A six-inch space is allowed between the two mattresses. The first blanket is placed squarely over the mattress, the next five blankets are folded lengthwise, then a rubber sheet three feet by nine feet in area is spread across the bed on the division; a blanket folded three-

corner style is placed at the head of the bed for the shoulders.

One large terry sheet is now spread. Before the patient is wrapped in this sheet the elbows, ankles, heels and coccyx are protected with absorbent cotton. After the terry sheet has been fastened securely, the rubber sheet is folded around the patient and the shoulder blanket is adjusted; four blankets, folded lengthwise, are then placed on top of the patient, and the first blanket is fastened securely with a safety pin, so as to form an "envelope". Three turns of the cable are now wound round the "envelope" and the machine is turned on.

By way of general preparation, two hours before the commencement of treatment the patient is given a hot bath, a salt and water enema, and ten ounces of hot milk or coffee. Also "Sodium Hebaral" (six grains) is given two hours before the treatment is commenced.

The pulse rate, respiration rate and temperature are recorded every fifteen minutes. Hot water containing glucose (10%) and saline (0·6%) is given freely during treatment. The temperature must be kept above 103·5° F. for eight hours, for two of which it is between 105·8° and 106·2° F. It is not advisable to raise the patient's temperature above 105° F. for the first two treatments. At the first treatment the machine is turned off at 103° F.; at the second treatment it is turned off at 103·4° F.; at the third treatment it is turned off at 104° F.

Some patients seem to acquire resistance to heat. Morphine (one-quarter of a grain) and hyoscine (one one-hundredth of a grain) are generally administered when the temperature is about 102° F.

We now take the blood for examination during the course of treatment, and should the Wassermann reaction disappear or be much diminished, the cerebro-spinal fluid is examined. Should the condition of this also have improved, no more heat is applied; otherwise the treatment is continued up to seventy or eighty hours (Prior, 1937).

We think we can now classify the results of treatment of 52 neurosyphilitics as follows: condition much improved, 21; condition improved, 22; condition unimproved, 9. Thirteen of these patients have died within five years of treatment.

Penicillin Therapy.

The action of penicillin is to cause excessive growth of the spirochæte leading to its death, according to experiments *in vivo*.

Although the passage of penicillin into the cerebro-spinal fluid is doubtful, there is no proof that an effective concentration of any chemotherapeutic agent does not occur in the tissues of the central nervous system.

The dosage has risen from two mega units to four or even six, given intravenously every three hours in doses of 50,000 units for a period of seven to ten days. These are trying to the patient, and latterly procaine penicillin in doses of 300,000 units has been given daily for fourteen days, a total dosage of 4·2 mega units.

Rarely febrile reactions accompanied by convulsions occur; these phenomena are regarded as Herxheimer reactions (Nicol, 1948). We commence with smaller doses of penicillin to reduce the liability to Herxheimer reactions—malaise, swelling of lymph glands and elevation of temperature to 103° F.

Nicol concludes that for early or late asymptomatic neurosyphilis penicillin therapy is probably adequate, provided a careful follow-up is observed. In late symptomatic neurosyphilis it is not recommended as replacing fever therapy.

Penicillin Given Intrathecally.

The recommended intrathecal doses of penicillin are 5000 units increased to 20,000 units at weekly intervals. The method used is to withdraw 35 to 45 millilitres of cerebro-spinal fluid into a cylinder, add the penicillin solution, raise the cylinder and allow the mixture to flow back into the spinal theca. This treatment is used for those who fail to respond to malarial or other therapy or if malaria is contraindicated (Thrasher, Gosman and Bowman, 1948).

Effects of Penicillin on the Cerebro-Spinal Fluid.

With penicillin treatment the cell count is normal by the end of two months. The total protein content may increase for a while, owing to reaction that has gone on within the cerebral tissue, and then become normal in six months—the globulin content becomes normal in six months. In most instances the Wassermann reaction ceases to be obtained in a year, while the colloidal gold reaction of paretic type may remain resistant to change for eighteen months to two years. As is well known, this persistence of the colloidal gold reaction also persists after malarial therapy and its significance is difficult to understand. It seems unlikely that the presence of the colloidal gold reaction should be regarded as a sign of the active persistence of the disease, although patients with such a phenomenon must be watched.

Thus the sequence of changes after successful penicillin therapy is exactly the same as that which follows successful treatment with malaria, and time will prove whether Dattner's insistence that cerebro-spinal fluid which has ceased to react to tests for syphilis after malaria never reacts to them again also applies to penicillin therapy (Martin, 1948).

Evaluation of Treatment.

It is considered that a period of five years is the minimum follow-up period for evaluation of any form of therapy. First reports were too optimistic. At present it is too early to give final answer to the question of the effectiveness of penicillin alone in neurosyphilis. There is still difference of opinion as to the wisdom of combining fever therapy with penicillin.

Thus in *dementia paralytica* 60 patients were given 25,000 units of penicillin three-hourly for a total of 6,000,000 units and 40 were given the same dosage of penicillin as well as induced malarial fever; no significant difference in results was noted.

In tabes 54 patients given malaria therapy combined with penicillin showed a clear-cut superiority of results to those obtained by penicillin treatment alone (Rose, 1949).

Spinal fluid examinations form the most reliable basis for evaluation of therapy in all types of neurosyphilis. Cell counts and total protein determinations are indicative of activity or inactivity of the syphilitic process within the central nervous system. Although some authorities, notably Grover, of the Veterans' Administration Hospital, Arkansas, state that the trend of the spinal fluid Wassermann reaction is a more accurate indication of parenchymal activity in paresis than cell and protein contents, and a more accurate prognostic guide, most other authorities at the present time do not support this statement.

In studying the clinical effects of psychotic patients with neurosyphilis it becomes increasingly evident with the passage of time following treatment that psychiatric symptoms alone cannot be utilized as a measure of failure or success of a treatment which is directed towards the spirochætal progress. This is indicated by improvement in 25% of patients with schizophrenic-like syndromes as compared with 90% improvement in patients with manic-depressive psychoses. This is further exemplified by experience in the use of electro-shock therapy in the psychosis of general paresis.

The most recent assessment of results of all types of therapy indicates that the type of psychosis present prior to treatment is of value prognostically. Psychosis of the effective type has an excellent prognosis; psychosis in the demented group has a fair prognosis; psychosis in the schizophrenic-like group has the poorest prognosis (London *et alii*, 1949).

Conclusion.

While any one or any combination of the above-described treatments—penicillin therapy, fever therapy or the administration of pentavalent arsenicals—may cause arrest of the pathological process within a year or more, the lack of associated clinical improvement in some of these

patients is due to (i) the neurological residuals of the disease and (ii) the type of psychosis present at the time of treatment.

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THE SCOPE OF GENERAL PRACTICE.[1]

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THE present time, when the Government has commenced its campaign to absorb the medical profession, is an opportune time to discuss the scope of general practice.

Let us start with some definitions. By "specialist" I mean a medical practitioner who confines his practice to one specialty or to several related specialties; a "consultant" is a specialist who confines his practice to patients referred to him by other medical practitioners; a "general practitioner" is a medical practitioner who does not confine his practice to one specialty or to several related specialties; and a "family practitioner" is a general practitioner who undertakes domiciliary practice.

In any large and under-populated country the importance of the general practitioner in the medical service is paramount. One obvious factor which restricts specialists more than general practitioners is the geographical consideration of population density; whereas a specialist can practise only in a populous centre, the general practitioner can be almost everywhere.

At the outset I wish to state that the scope of general practice is all medical practice. This does not imply that there is—and there certainly should not be—a conflict between general practitioner and specialist; many occasions arise when special knowledge and skill, possessed only by one who confines his practice to a limited field, must be sought.

In a particular case the scope of general practice, as also of any medical practice, is determined by the application of the golden rule; in the existing circumstances and conditions would the practitioner, if he were the patient, be treated by him in the way recommended? However, in answering the question to himself the practitioner is not expected to assume the absolute self-confidence required of a successful national war leader.

One of the greatest attributes which any medical practitioner can possess is a knowledge of his own limitations, and the competent practitioner will know when and where to seek the assistance of special knowledge and skill and of mature judgement.

As medical knowledge increases, the need for specialization also increases; nobody can hope to be sufficiently skilled or to have the necessary equipment to undertake all investigations and treatment.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on October 7, 1949.

The general practitioner, however, must not only be able to diagnose and treat many patients who are ill, but also he must know what necessary investigations and treatment are available by other practitioners in special branches.

In a society based on the family unit the basis of medical practice must be the family practitioner, and in Australia, where there are many small isolated communities, it is of primary importance that many general practitioners capable of dealing with all medical needs should be available.

Whilst agreeing that a family practitioner should take every opportunity of advising patients in the prevention of disease—and I think much preventive medical advice is given by family practitioners as part of their daily routine—I do not agree that the general practitioner should become a health visitor whose duty should be largely that of advising on personal hygiene and nutrition. Sir Farquhar Buzzard, in his Harveian Oration for 1941, suggests a limitation of general practice in this way.

Conditions in the United Kingdom differ from those in Australia, so that opinions expressed regarding the scope of practice in one country may not apply to that of another.

If we must make a general statement regarding the duties of general practitioner and specialist, I should say as a partly true generalization that the specialist treats diseases and the general practitioner treats patients.

With all medical practice as the general practitioner's scope, it is necessary to ensure his competence. Every opportunity must be offered to improve his knowledge and skill and to expand the scope of his activities so as to keep pace with medical progress, and your attention is again invited to the fact that the first objective of the British Medical Association is the dissemination of knowledge of medical science. The goal at which the Royal Colleges and the special groups aim is not the prohibition of special types of practice by non-members, but the encouragement of medical practitioners to acquire special knowledge and skill and, as far as possible, to offer facilities to enable them to acquire it.

The training of general practitioners must be the principal objective of our medical schools and teaching hospitals, though it is unfortunate that inadequate finances and staff at present prevent them from providing post-graduate training and from undertaking research on a scale which they otherwise could and should do; these activities stimulate teachers and undergraduates.

It is a serious misfortune to the community that the members of the staff of a medical school should be so fully occupied in undergraduate teaching that they can have little time for reading current medical literature, much less for undertaking research and post-graduate teaching.

However, I am amazed not at the small number of full-time medical teachers, but at the high attainments of those few when much more lucrative activities would be easily available to them.

Undergraduate training should provide a sound basis upon which to build a good knowledge of all branches of medical practice, and our Australian universities strive to give this as well as their facilities allow. There will always be differences of opinion about the details of undergraduate training; whether minute details of anatomy, abstruse biochemical problems and long discussions on rare diseases should be inflicted on the undergraduate will always be controversial. I am not competent to express an opinion, nor can I indicate what, if anything, should be omitted to relieve the undergraduate's burden.

Undergraduate training for a profession which is continually advancing must also change and improve continually.

It is doubtful whether the medical course is now more onerous than it was thirty years ago; recent graduates seem to be at least as well equipped for their duties as were those of former years, and they appear to have suffered no harm from the alleged hardships of the present medical curriculum. It is easy for critics to lose perspective when pleading for their particular hobby or when condemning their own aversion.

It is apparently inevitable that most clinical teachers should be specialists and that some of them tend to overemphasize the importance of their own specialty, as though all students would eventually practise that specialty. However, this tendency is obviously preferable to a lack of enthusiastic interest. It is also unfortunate that some very competent specialists in teaching hospitals devote too little time and thought to making themselves good teachers.

Ethics is a subject upon which sufficient and correct emphasis is not always placed. Not only should special lectures be given on medical ethics, in which the duty of the medical practitioner to his patient, to the State, to his colleagues and to himself is indicated, but all clinical teachers should emphasize the practitioner's important duty to offer his patient the best available means of overcoming his disability, which may require another opinion or the transfer of the patient to another practitioner; and also his duty to his colleagues and patient to avoid undermining a patient's confidence in another practitioner.

With regard to the practitioner's duty to his patient, it should be remembered that a patient seeking advice does not therefore require the practitioner to undertake necessary treatment which he is not competent to give or for which he has not the facilities.

With regard to the practitioner's duty to his colleagues, confidence in his medical adviser plays a definite part in assisting a patient's recovery, and anything which tends to undermine that confidence is harmful. Not often, but too often, one hears of patients who have been informed how unskilfully they were previously treated; this is a very serious ethical breach.

A term as resident medical officer is essential for every medical practitioner, and during that time he requires the widest available practice in sound medical methods and should be trained to deal with all emergencies likely to be encountered if he practises in an isolated community. Watching a procedure is not sufficient training, and it is the duty of the staff of hospitals with resident medical officers to ensure that all residents have opportunities to carry out all emergency procedures under supervision.

The casualty and admission rooms and the out-patient clinics of a hospital offer excellent material for training general practitioners, and their importance is not sufficiently emphasized. Too often do recent graduates consider that work in these departments is beneath their serious notice as future leaders in their profession. Too much emphasis and glamour have been attached to the care of patients with established diseases who require spectacular treatment; but the treatment of patients with minor ailments and the recognition and care of early manifestations of disease provide the major portion of medical practice. Enthusiastic specialists on the teaching staff must bear some of the blame for this superior attitude to the casualty room and related clinics; but hospital administrators should ensure that a senior practitioner is in charge of these departments, so that proper training will be available and its importance realized; such a senior practitioner would probably be able to coordinate the knowledge of the various specialties and to give the resident medical officer and the undergraduate a better perspective of medical practice.

The desire of the Queensland Government a few years ago to require the serving of a term as resident medical officer before full registration as a medical practitioner was excellent, and it is regrettable that reciprocity agreements caused this qualification to be postponed until other registration authorities become sufficiently enlightened to require such a training.

Senior resident appointments give opportunities for commencing the training of specialists and for the more brilliant students to prepare themselves as candidates for higher degrees and diplomas.

I think, however, that some experience as a family practitioner is an essential part of the training of every specialist; this experience gives him a knowledge of the environment of patients which he cannot otherwise acquire, and it may even help him to be more tolerant of his colleagues.

Post-graduate reading, lectures and demonstrations and contacts with colleagues are as important for general practitioners as for specialists. It is therefore desirable that general practitioners should have better access to private, intermediate and public wards of hospitals.

In small towns all general practitioners should have the opportunity of securing part-time appointments to the public hospital, and in larger towns many clinical assistant appointments should be available to general practitioners for a year at a time, so that a large number could enjoy the contacts with professional colleagues and the experience available at the clinics. The critical discussion of cases with colleagues is a useful part of post-graduate education and is usually possible only on the staff of a hospital.

The difficulties of general practice are at present aggravated by the lack of domestic help and the scarcity of suitable *locum tenentes*.

Some form of group practice and the arrangement of a roster of duty by neighbouring practitioners are methods of helping to overcome the great difficulty of providing a twenty-four hours a day service.

In a one-man town the practitioner carries a heavy burden, and he needs every assistance to ensure regular holidays and opportunities for refresher courses.

The time of clinical meetings and scientific discussions could be varied to discover what is suitable to most practitioners; anyone who has evening hours finds it very difficult to reach a meeting by 8 o'clock.

The future of general practice is at present a matter for conjecture. Two factors will have a great influence on it. First there is a real need for financial assistance to patients who are faced with unexpected heavy medical, hospital or pharmaceutical expenses, and governments all over the world are making efforts to solve this problem in various ways. One obvious way of affording help would be to reduce considerably the present heavy direct and indirect taxation, and so leave the individual sufficient income to meet such expenses; but this method does not appeal to those governments which are intent on extending paternalism and controls to the exclusion of individual enterprise and freedom, and which demand an enormous revenue for the alleged purpose of providing social security for everybody. The plea for social security, free medicines and medical services and full employment is fashionable in political circles, but it should be remembered that the prisoner in gaol enjoys these privileges. Financial assistance for the occasional visit to a doctor or for occasional medicines is not required by the great majority of families, and it is the Government's attempt to thrust this unwanted assistance on the community that will cause almost all the administrative difficulties and much obnoxious interference with medical practice. My own opinion is that financial assistance for medical expenses should be given only after the patient has paid a certain amount in any one year, just as with many motor-car insurances the insured person is responsible for the first five pounds or some other small amount. Special arrangements should, of course, be made for pensioners and unemployed persons. In the case of medicines, I think the suggestion which Dr. A. J. Collins made to the Minister for Health and Social Services at a conference with the Federal Council is the best solution—that is, to make all the specific drugs free to the patient; this could be done by providing these drugs free of charge to the pharmacist and paying him a storage and a dispensing fee.

The second factor which will influence the future of general practice is the increasing tendency of governments to extend State control and to diminish individual effort and freedom. Governments are not now willing merely to determine and enforce standards; they wish to control as many activities as possible, and this desire does not appear to be the prerogative of one party. Whether the Government will be persuaded to assist in meeting heavy medical expenses without unnecessary interference with the sound basis of medical practice or whether it will continue to aim at a complete full-time salaried service will determine the ultimate scope of general practice.

If medical practice continues as a private enterprise more facilities for general practitioners to accept hospital appointments are required, and closer cooperation between neighbouring practitioners and possibly more group practices appear to be desirable.

If governments continue to encroach on medical practice by subjecting it to more and more departmental control, then the scope of general practice will shrink, and in proportion to the reduction of individual initiative the standard of practice will be lowered to a poor mediocrity.

Government control will not only tend to bring all practitioners to one level—and unfortunately the level is likely to be that of the least enlightened practitioner—but it will encourage the practitioner to avoid responsibility, and the departmental worship of the god of seniority will tend to remove the incentive to improve the standard of practice.

One may think about government control of medical practice what one cannot avoid thinking when one looks at some examples of modern art—that things cannot be as bad as they are painted; but excessive control and standardization are serious checks on individual effort, and medical practice is so largely individualistic that it is hampered more severely than many other services.

Fashions change in medicine as in other things. Only a few years ago many were of the opinion that the day of the general practitioner had passed and that most medical care would soon be provided at hospitals by specialists. This opinion was held not only by our uninformed politicians but also by many of our medical colleagues. At present the emphasis is on the general practitioner as the foundation of medical practice, and fears now frequently expressed by medical authorities are of over-specialization and of too restricted specialties.

The suggestion in recent years that special departments of social medicine should be established in medical schools is an acknowledgement of the importance of the family practitioner in medical practice.

No inflexible rule can set out the scope of practice for all practitioners. A competent practitioner will give such advice and treatment as his training and experience permit, and will refer to other practitioners with special knowledge those problems which he himself cannot expertly solve.

Training, experience and existing facilities may enable a general practitioner to perform much excellent work in a particular specialty without losing touch with other branches of medical knowledge; and the ideal specialist is one who not only excels in his own specialty, but also has sufficient knowledge of other branches of medicine to be a good general practitioner.

In an unfettered medical profession such competent practitioners are frequently met, and private practice offers the best incentive to achieve competence. A serious fault in a government-controlled service is that regulations are made which determine too rigidly the scope of each individual's practice and thus stifle initiative.

No method of training and no method of control will achieve perfection, but training and control should continue to aim at competence.

The best medical service for the community will be achieved by ensuring that competent private general practitioners are available to all members of the community; competent general practitioners will have a sound knowledge of a wide field of medical practice and will be capable of dealing with all medical emergencies, but should constantly apply the golden rule to their practice, as also, of course, should specialists and even members of governments.

Summary.

To sum up, the scope of general practice is all medical work which the particular general practitioner can competently carry out.

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ANTE-PARTUM AND POST-PARTUM HÆMORRHAGE.[1]

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ANTE-PARTUM HÆMORRHAGE.

THE three outstanding causes of maternal mortality are (i) infection, (ii) toxæmia, and (iii) hæmorrhage.

Infection used to be far and away the most frequent cause of death, with toxæmia next in importance. During the last two decades improved prophylaxis in ante-natal care, improved technique and better conduct of labour—due probably to the steady increase in the number of doctors specializing in obstetrics, to the increasing influence of the Royal College of Obstetricians and Gynæcologists, and to the introduction of chemotherapy—have produced a steady reduction in the mortality and morbidity in both infection and toxæmia.

In most countries now hæmorrhage is the most frequent and most important cause of maternal death, and post-partum hæmorrhage is responsible for more deaths than ante-partum or intra-partum hæmorrhage. Many deaths classified under "ectopic pregnancy", "abortion", "rupture of the uterus" are primarily due to hæmorrhage, while a large percentage of deaths classified under "infection" have been caused by hæmorrhage, and the efforts to control hæmorrhage, which have reduced the patient's resistance to infection and so have been primarily responsible for death.

It should be more widely recognized that a large percentage of deaths from hæmorrhage are preventible. An important factor is the recognition among obstetricians of the importance of replacing blood lost before, during or after labour. It is unnecessary for me to point out the inestimable value to the community of the Red Cross work in organizing blood banks. Only the older practitioners can really appreciate the great blessing to doctor and patient of this service. Two decades ago we had a large percentage of maternity cases in private homes, assisted by trained or untrained "Gamps". We had no commercial preparations of saline or glucose solution and had to prepare our own. For blood we collected relatives and friends of the patient, and cross-typed specimens till we found a compatible donor—and of course we did not know the cause of the bad reactions we sometimes met with, knowing nothing of the Rh factor.

Now, with a large percentage of obstetric patients delivered in hospitals, with the steady improvement in hospitals and staffing and the gradual elimination of the small so-called private hospitals, with universal blood banks and routine ante-partum blood grouping and Rh factor determination, we should be able to reduce the mortality from all causes, but especially from hæmorrhage.

I have no time to discuss hæmorrhage in early pregnancy—usually abortions and ectopic pregnancies—and can only breifly refer to the main causes of hæmorrhage in late pregnancy. These are (i) accidental hæmorrhage or *abruptio placentæ* and (ii) *placenta prævia*. Since it is not possible to make an accurate diagnosis as to the type at the first hæmorrhage, which as a rule is not severe, it is imperative that all patients with hæmorrhage in the last trimester be sent to hospital at once. A gentle examination of the cervix and vagina can safely be made with a speculum when the patient has been admitted to hospital, to exclude local causes of bleeding, such as a cervical polypus, cancer *et cetera*. though even if a polypus is found one cannot with certainty exclude the possibility of placental bleeding.

Accidental Hæmorrhage.

The differential diagnosis between the two types of ante-partum hæmorrhage is not usually possible without

[1] Read at a meeting of the Section of Obstetrics and Gynæcology, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

digital examination of the cervix or X-ray investigation, or a combination of both. Most of us would agree with the following points: (i) It is not possible to differentiate between accidental hæmorrhage and *placenta prævia* hæmorrhage on the character of the hæmorrhage alone. (ii) The diagnosis of *placenta prævia* should not be made unless the placenta has been felt in the lower segment, or after operation. In some cases an X-ray examination may justify making a diagnosis. (iii) Only when the type of *placenta prævia* has been determined can one decide on the best method of treatment. I have found the cystogram (Ude and Urner) of diagnostic value in certain cases, especially when a normally situated placenta is present. A negative result is not helpful, as the placenta may be in the pelvis.

Typical clinical manifestations of accidental hæmorrhage are threefold: (i) continuous uterine pain—especially if it is a concealed hæmorrhage; (ii) tenseness and tenderness of the uterus, especially locally at the site of the separation (this local tenderness, I have usually found, persists to the time of delivery); (iii) external hæmorrhage, with or without signs of concealed hæmorrhage.

Treatment.

In mild cases admission to hospital and no interference are sufficient if mother and baby are not showing distress. In more severe cases, when the hæmorrhage continues and cannot be controlled, replace the blood lost at once, treat shock (with oxygen *et cetera*) and empty the uterus as soon as possible, the method depending on the condition of the mother, of the baby, and of the cervix. Preparations are made for immediate blood transfusion when this is indicated; the patient's blood is grouped and the Rh factor is determined, if this has not already been done, and compatible blood is stored and ready for use. The patient is of course kept in hospital until she has been delivered—usually with complete bed rest.

In severe accidental hæmorrhage, the chances of obtaining a living baby are slight, particularly in the concealed or mixed varieties.

Expectant treatment is thus not often adopted, and the case is treated from the standpoint of the mother's safety only. Various methods of induction of labour have been advocated by different schools, but the simplest and most generally used now is surgical induction. Occasionally in certain cases hysterectomy or Cæsarean section may be the best treatment.

Placenta Prævia.

The treatment of *placenta prævia* depends on several factors: (i) whether the blood loss has been severe and is continuing, and on the condition of the patient (if she is in a state of shock *et cetera*); (ii) whether the baby is still alive, in which case conservative treatment may be justified, often depending on the age and parity of the patient; it is usually justified for elderly *primiparæ* or for women who have been married for many years and have had no children; (iii) the type of placental attachment; (iv) the degree of cervical dilatation if the patient is in labour.

Manual dilatation of the cervix must never be attempted in *placenta prævia*. If there is malpresentation of the fœtus, Cæsarean section is indicated. My treatment has been in line with that of others—that is, conservative until and unless hæmorrhage forces interference, with complete hospitalization and complete bed rest. All preparations should be made for immediate interference if necessary. When interference is necessary, if a definite diagnosis has not been made by X-ray findings, a vaginal examination is carried out in the operating theatre, with all preparations for an abdominal delivery if this is considered advisable, including preparatory blood transfusion if the patient's hæmoglobin value is below normal.

Treatment.

If vaginal examination proves the condition to be of Type III or Type IV (that is, partial or complete in Professor Francis Brown's classification), or of Type II (marginal), if the placenta is on the posterior wall (the antero-posterior diameter at the brim being thus reduced) and if the head is not engaged, a lower segment Cæsarean section is indicated. If the condition is marginal (type II) and the placenta is not on the posterior wall, or of Type I (lateral), a Cæsarean section is not considered necessary and induction of labour is the probable method of choice.

The position of the head is of importance in deciding the course of treatment. The engaged head is more likely to control the hæmorrhage than one above the brim.

If the cervix has taken up, and especially if contractions are present, a surgical induction is indicated, and if necessary, the application of Willett's forceps on the scalp will help to control any bleeding, but will increase the fœtal mortality to some extent. I have used this procedure with good results in some cases. The Braxton Hicks podalic version is, I think, seldom employed now unless the fœtus is dead. There is, of course, a risk of wide separation of the placenta and also of rupturing the uterus.

As to the type of Cæsarean section: of late years, if the patient's condition is satisfactory and the placenta is considered to be on the posterior wall, I perform a lower segment section with the transverse incision; but if the anterior wall is infiltrated with large vessels, and palpation makes it probable that the placenta is on the anterior wall, I prefer vertical incision—even raising the site and making the operation a low classical section. In the classical operation I find it easier to control massive hæmorrhage, if it occurs, by using a rubber tube tourniquet (Chambers) round the lower segment while sewing up the uterine incision and/or packing the lower segment with gauze, first passing the end well down into the vagina.

If the cervix is closed I use a small Hegar dilator, having the handle cut to the same size as the dilating end, with a hole in it through which the end of the gauze pack is threaded and tied. The dilator is pushed through the cervix well down into the vagina and can easily be picked up when the pack is removed.

Montgomery advocates packing the vagina tightly when the patient is examined prior to the Cæsarean section, and after the placenta has been removed the lower segment is tightly packed before the uterus is sutured, so that the lower segment is held tightly between two packs.

I suggest the following as an alternative. Pack the uterus from above at operation and then pack the vagina rapidly at the end of the operation. The lower end of the upper strip will be found in the vagina, pulled down and held while the vaginal packing is being done. This facilitates removal of both packs when required. "Oxygauze" may prove a useful packing agent in the lower segment when the bleeding is not severe.

POST-PARTUM HÆMORRHAGE.

Causes.

The causes of post-partum hæmorrhage are as follows: (*a*) uterine inertia; (*b*) retention of placenta or placental parts; (*c*) injuries—lacerations of the genital tract, including an episiotomy wound, if not inspected and controlled; (*d*) abnormal placental site or attachments; (*e*) improper conduct of any stage of labour, such as versions in the late second stage, the application of forceps with incomplete dilatation of the cervix, "manhandling" the uterus in the third stage *et cetera*.

Among the causes of uterine inertia we must recognize the importance of the condition of the patient at term and the importance of admission to hospital for a few days or weeks before term of all patients suffering from undernourishment, anæmia, over-tiredness from lack of home help and rest, over-distension from hydramnios or multiple pregnancy *et cetera*. Other causes are exhaustion from prolonged labour, ante-partum hæmorrhage in which lost blood has not been replaced, prolonged inhalational anæsthesia, and especially repeated anæsthesia with insufficient use of oxygen.

Prevention.

Adequate Ante-Natal Care.

I can only briefly refer to a few of the factors concerned in the limitation of this most important cause of maternal mortality.

Blood grouping early in pregnancy is becoming universally a routine factor in ante-natal care, and I need not emphasize its importance.

The nutritional state of the patient at term is of first importance for the sake of the baby and also for that of the mother, particularly if she has to go through a prolonged or difficult labour. I would emphasize that merely giving the patient a leaflet or book is not enough—one must repeatedly question her at her visits about how she carries out these instructions. When it comes to doing something patients do not like doing, a large percentage of them do not do it.

Routine hæmoglobin estimations should be made and when iron is given follow-up hæmoglobin tests are required. We must remember that because of hydræmia the normal hæmoglobin value in late pregnancy is lower than at other times. Blood counts should be made when indicated. The importance of adequate blood replacement in cases of ante-partum hæmorrhage must be recognized.

As the percentage of accidental hæmorrhage is high in toxæmias, special observation and treatment of all patients with toxæmia should be carried out, preferably in hospital. This should reduce the cases of accidental hæmorrhage.

Rest at home or in hospital should be insisted upon for over-tired or debilitated patients before the onset of labour.

Labour.

Stages I and II.—In Stages I and II of a prolonged labour a very important factor in the prevention of shock is adequate nourishment; patients who have acidosis are more likely to develop shock. We are inclined to leave this matter to the nursing staff and not to make certain that everything is being done to see that each patient receives individual attention. Patients under analgesics will not attend to this themselves and must be awakened at intervals and made to take fluids, glucose and light nourishment. It may be advisable in some cases to administer saline and glucose solution intravenously. The importance of a well-trained nursing staff in the labour wards must be recognized.

Fatigue and exhaustion must be combated by giving the patient adequate sedation, especially in prolonged labour. Professor H. L. Sheehan (1948), in his interesting address on obstetric shock, based on clinical study and post-mortem examinations in 147 fatal cases of shock in relation to labour at the Glasgow Maternity Hospital, emphasized the occurrence of acidosis in prolonged labour as a result of starvation, vomiting and long-continued muscular activity. All deaths in this series occurred among patients who had been in labour more than two days. Professor Sheehan also spoke a word of warning that these "starvation" patients are those who invariably develop delayed chloroform poisoning if they are given chloroform even in small doses.

Anæsthetics are probably contributory factors in the occurrence of hæmorrhage and shock. Inhalational anæsthesia—especially with ether—if excessive, causes undue relaxation of the uterus. In the terminal stages of delivery, for normal patients I prefer chloroform if the anæsthesia is short, especially if hyoscine has been used; ether tends to excite such patients and they expend much muscular energy and have to be given an excessive amount of ether to control their struggles. However, chloroform, as Professor Sheehan points out, should never be used in cases of prolonged labour in which acidosis is present.

Harold Bishop (1947) reported the occurrence of four sudden deaths due to the use of pituitrin in the treatment of patients under cyclopropane anæsthesia, as they were passing from deep to a lighter stage of anæsthesia. Cyclopropane is becoming more popular as an anæsthetic agent, especially in Cæsarean section, and pituitrin is used more or less as a routine measure before the uterus is opened. Although a discussion on anæsthetics is outside the scope of this paper, both cyclopropane and pituitrin may be used in cases of post-partum hæmorrhage, and I feel impelled to draw attention to this report.

With regard to the conduct of labour and meddlesome obstetrics, I need do no more than refer to the possible risks attending unnecessary or premature interference in any stage of labour, or to the dangers connected with the misuse of pituitrin in the induction of labour or in cases of secondary uterine inertia. Much has been written, especially in journals in the United States of America, on the occasional occurrence of pituitrin shock and of rupture of the uterus following pituitrin injections. I have encountered two cases of rupture in which pituitrin was used for induction of labour. One might mention also the misuse of forceps as a factor in post-partum hæmorrhage—for example, too early application, before full dilatation of the cervix, rotation of the head with forceps by unskilled obstetricians, or delayed application and a too prolonged second stage, the patient being allowed to become unnecessarily exhausted.

Stage III.—In Stage III passive control of the uterus should be observed in normal cases. Most of us condemn the use of the Credé manœuvre while the placenta is still *in utero*, because of the potential dangers of "mashing" the placenta and the risk of retention of these separated portions, hæmorrhage and subsequent infection. If Credé expression is used before the placenta has separated and when it is not in the lower segment or vagina this criticism is justified because, roughly performed, especially if repeated—the manœuvre can be the cause of profound shock. Used to express forcibly or to squeeze the placenta out of the uterus, it can be dangerous. Gentle traction on the cord may be used when the placenta is in the lower segment or vagina and the uterus in contraction. Downward pressure on the uterus with the other hand will usually be sufficient to expel the placenta or, perhaps better still, upward traction on the uterus with the hand above the pubes.

Oxytocics.—Pituitrin and ergot derivatives are coming into more general use at the end of the second stage of labour. I have no time here to discuss the advantages and possible disadvantages and dangers of this practice. Chassar Moir, Dieckmann and other authorities sanction their use by experienced obstetricians only. I have for many years used pituitrin intramuscularly as the head is delivered, and of late have used as an alternative the intravenous injection of ergometrine (0·25 milligramme) as soon as one shoulder is delivered. The former is, I consider, a safe and useful procedure; but I have a reserve in my mind about the latter. I have had several cases of ring contraction and prolonged tonic contraction with the placenta still *in utero* that caused considerable trouble after the intravenous administration of ergot, and we may yet return to our former teaching that to give ergot before the placenta is delivered is almost a crime.

Dieckmann and others have emphasized the importance in the second stage of allowing spontaneous delivery of the body after the head is delivered. This may take three or four minutes and gives the uterine wall time to contract and retract, thereby tearing itself away from the placenta and shortening the third stage.

Cord Injection.—About ten years ago I began to use injection of the umbilical vein with normal saline solution at blood heat for retained placenta. I found it of value and I then experimented with the temperature of the solution and found that the temeprature fell about 10° F. in passing along a foot of the cord and out of the placenta. I then tried hot saline solution (from 120° to 128° F. if I was using a foot or more of the cord), and employed a large-bore needle or nozzle in the syringe, so as to enable the injection to be given rapidly.

I consider that the hot solution and the rapidity of injection are both important factors in stimulating contractions. I first used this treatment for retained placenta and in a high proportion of cases it proved of value—84% success, as will be shown later. However, I now place more emphasis on its value in arresting hæmorrhage, particularly when there is a steady ooze from an atonic uterus. Besides stimulating contractions and arresting hæmorrhage, it will in many cases spontaneously expel the placenta during the injection or allow it to be expressed immediately or soon after the injection has been completed. It is useful prophylactically in cases of intrauterine inter-

ference in the second stage, such as manual rotation, "high forceps" delivery or previous manual removal or hæmorrhage in previous deliveries, and I often use it immediately after the baby has been separated in such cases. It would not, of course, be used in cases of severe post-partum hæmorrhage, when the only course would be an immediate manual removal. In cases in which the placenta is retained after a cord injection and a manual removal has to be carried out, one has the advantage of handling a thick, engorged placenta, and it can often be grasped and pulled out complete, with the hand in the vagina or lower segment only.

This method will not effect delivery of the placenta in cases of hour-glass contraction or *placenta accreta*, but will frequently check the hæmorrhage, and in the former cases it often allows natural removal of the placenta later when the ring relaxes. It has, of course, no effect on the uterine contraction if all or most of the placenta is in the vagina—retained by a lower contraction ring grasping a small section of the placenta. When the solution begins to run out of the vagina soon after the injection is started this is usually the reason, and of course little if any of the saline enters the uterus.

To sum up: it is a safe and easily given hot intra-uterine douche which reduces the amount of blood loss, lessens the chance of infection and stimulates uterine contractions, so that the placenta is frequently expelled. If not, it can usually be expressed soon afterwards, and even if the placenta is still retained by a contraction ring no harm has been done. On a few occasions in such cases I have carried out the injection a second time successfully about twenty minutes later.

The following details are given of 182 cases in 3009 consecutive confinements in which the cord injection was used for uterine inertia and hæmorrhage in the third stage, or when this stage was unduly prolonged. Of these 182 cases 50 (27·4%) were very successful (the placenta was spontaneously delivered before the injection was completed), and 104 (57·1%) were successful (hæmorrhage checked and placenta delivered within a few minutes)—a total of 84·5% successes. In 28 cases (15·5%) the method failed—the placenta was retained up to one hour; in 10 of these cases the placenta had to be manually removed (0·3%). Three of these patients had had previous manual removals of the placenta, and four had contraction rings. In 1350 previous cases I had performed 18 manual removals of the placenta (1·3%).

I have made a short film of this simple procedure. I feel sure that if hospital delivery wards had the simple apparatus autoclaved, it would often be used and its value in suitable cases would be appreciated.

Manual Removal.—Manual removal of the placenta is not the serious undertaking it was before the introduction of chemotherapy, and when a large percentage of our obstetrics was carried out in private homes or in small private hospitals, without proper asepsis and without oxygen cylinders, blood for transfusion and other methods of dealing with patients in a state of shock. It was as a rule resorted to only when hæmorrhage had been so severe or the placenta had been retained so long that no other course was open to the doctor.

We now seem to be going to the other extreme and many of the American clinics are advocating manual removal almost as a routine measure, within a few minutes after birth of the baby. American journals publish results which are apparently satisfactory—no mortality and a low morbidity rate. One cannot help recalling the fact that our American cousins in the early thirties showed the same enthusiasm for using Cæsarean section as an easy solution for their obstetric problems, and the maternal mortality rate rapidly increased.

If manual removals become generally adopted more or less as a routine measure I feel that the maternal mortality and morbidity rates would show a similar steady rise. DeLee states that "performed as it usually is, it is more dangerous than most laparotomies". This operation can be reasonably performed by skilled obstetricians in first-class hospitals, and I agree that it has to be employed if hæmorrhage is massive, persistent and uncontrollable, or if the placenta is retained over one hour or at the most two hours. Professor Sheehan (1948), in his address on shock in obstetrics already referred to, emphasized the shock danger in the third stage, which has lasted over two hours.

One feels, however, the value of conservatism in general. If a simple measure such as the cord injection, which does not require more than ordinary care and skill, is going to reduce the number of such invasions of the uterus and if a proper conduct of labour in all stages is strictly adhered to, the patient's best interests will be served. For the cord injection the only condition requisite is that the delivery ward should have the simple apparatus ready sterilized for immediate use at any time.

Packing the Uterus.

Use of the uterine pack for hæmorrhage after the placenta has been delivered is not often referred to in British text-books or journals, but many American obstetricians advocate it and some enthusiastic articles have recently been published in American journals.

Dieckmann uses it if bleeding cannot be controlled by the intravenous administration of oxytocics, by manual exploration of the uterus and by massage through the abdominal wall. Greenhill gave up using packing long ago. Cosgrove and others consider that it is unphysiological, that it holds open the sinuses, allowing bleeding to go on, and that the uterus then distends rather than contracts, and the hæmorrhage is converted into the concealed variety.

Most British text-books also view the method with disfavour.

Bimanual Compression.

Hamilton in 1861 first advocated the use of bimanual compression as a method of controlling late post-partum hæmorrhage, and it is still commonly used in Great Britain for severe post-partum hæmorrhage. Professor Eastman, of the Johns Hopkins Medical School, states that it has many points in its favour and he cannot understand why it has been almost entirely neglected in the United States. The clenched fist in the vagina rotates the knuckles against the anterior wall, while the external hand massages the posterior wall and compression of the uterus between the two hands occludes the uterine sinuses. Eastman points out that the method (*a*) provides massage for both walls of the uterus, (*b*) requires no invasion of the uterine cavity and lessens the risk of infection, (*c*) requires no special equipment, (*d*) has proved efficacious in the hands of countless obstetricians.

Late Post-Partum Hæmorrhage.

Late post-partum hæmorrhage occurring after the first day is usually due to retained fragments of placenta or to a *placenta succenturiata*. These may cause sudden and alarming hæmorrhage as they partially separate. These cases must be dealt with at once by digital exploration or a curette, and blood replacement must be effected as soon as possible.

Conclusion.

Finally, I would emphasize the importance of keeping a careful watch on the uterus for at least an hour after the placenta has been delivered—in what Greenberg calls the fourth stage of labour. Hæmorrhage may be concealed or revealed, and the uterus must be palpated and the vulval pads inspected frequently. I always give ergot by intramuscular injection immediately the placenta has been delivered in all cases in which ergometrine has not been given intravenously as the baby was being delivered.

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THE CLOSED HEAD INJURY: MODERN CONCEPTIONS AND THEIR INFLUENCE ON TREATMENT.

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IN the past ten years there has been a remarkable revival of interest in problems associated with head injuries. Primarily, these problems were the concern of the general surgeon and practitioner, and it is true that in large measure they must remain so, in spite of the recent development of neurosurgical centres in all larger hospitals. But during the war years neurosurgeons obtained mass experience in traumatic cranial surgery, and many have found it a satisfying and fascinating field for organization, research and endeavour. The tremendous expansion of physiological research into the central nervous system has extended into traumatic conditions. Nor has the pathologist been idle in this renaissance. On the economic side also, great advances were made in the organization of treatment in special centres throughout the armed forces. In peace time, though the majority of patients with head injuries will be cared for by the general surgeon or the general practitioner, some organization must be developed to deal with "problem cases".

FIGURE I.
The acceleration injury.

With the rapid development of knowledge it is obvious that conceptions must change, and that these new conceptions must modify, not only the treatment of head injuries, but also the teaching of the subject to students. The teaching on head injuries still tends to centre around the conception that concussion, contusion and cerebral compression are three clinically distinguishable states, and from this premise a number of misconceptions have occurred and treatment has tended to become inactive for too long. There is now a tendency to refrain from active treatment until the signs of compression are obvious. By this time it is often too late for any active procedure to be of any avail. The frequent failure of active treatment at this stage has led to the conclusion that operative treatment is rarely justified and that one must be satisfied with the results of conservative treatment. In reality this has meant being satisfied to do nothing or to make little more than a half-hearted attempt at dehydration. This principle appears to have been built up on concepts and premises which, although not yet discredited, seem uncertain in the light of new knowledge which has been obtained. It is proposed to review some of these new concepts briefly before further consideration is given to the management of the blunt head injury in the early stages.

Physical Concepts.

In recent years much work and considerable ingenuity have been expended on the investigation of the mechanics of head injury. Much of this work has centred around the nature of concussion. Although old concepts have been revived, the recasting of ideas by various investigators has stimulated thought and introduced modifications in treatment.

Much of the recent work followed a paper by Denny-Brown and Russell (1941), who stated that concussion is a phenomenon which is directly related to acceleration or retardation effects on the brain itself. In doing so they define two types of injury: (i) the acceleration type (Figures I and II), usually associated with the phenomena of concussion; (i) the compression type (Figure III), which, in spite of a high and rapid increase of intracranial pressure, is not necessarily associated with unconsciousness. In the acceleration type of concussion, they could detect no characteristic macroscopic or microscopic change in the involved brain, and they postulated that the phenomena of concussion were due to some physical injury on the neurons themselves.

Holbourn (1943), a physicist, carried this work a stage further. According to his theory, rotation of the head sets up shear strains in the cortex and underlying white matter. These shear strains pull the constituent particles of the brain apart, and it is this phenomenon which is the essential feature of brain injury. Since concussion occurs only when the head is subjected to a change in velocity, he suggested that a rotational movement takes place between the skull and brain and that the rotational shear strains produced are the cause of concussion.

Although not supporting the view that concussion is due to such rotational injuries of the cortex, R. D. Wright (1946) agrees that the most important factor in the

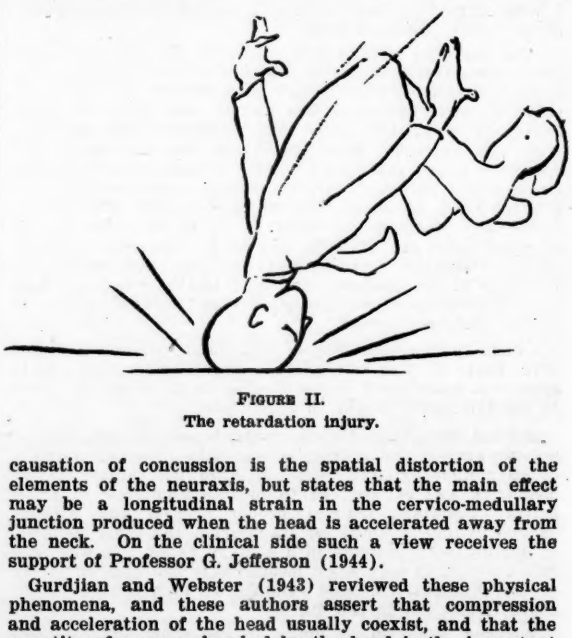

FIGURE II.
The retardation injury.

causation of concussion is the spatial distortion of the elements of the neuraxis, but states that the main effect may be a longitudinal strain in the cervico-medullary junction produced when the head is accelerated away from the neck. On the clinical side such a view receives the support of Professor G. Jefferson (1944).

Gurdjian and Webster (1943) reviewed these physical phenomena, and these authors assert that compression and acceleration of the head usually coexist, and that the quantity of energy absorbed by the head is the important factor in producing concussion. With a cathode ray oscillograph they have shown that the brain is subjected to a high initial pressure wave followed by a few rapidly damped oscillations. It is reasonable to assume that these disturbances will injure the cells and supporting structures in direct proportion to their intensity. The apparent concentration of such waves at the cranio-spinal junction may explain the invariable involvement of the vital centres in this region.

The macroscopic lesions—contusions and lacerations of the cerebral cortex—were studied by Holbourn (1943), who suggested that these were more frequently due to rotational forces within the cranium than to the accepted mechanism of *contre coup*. Holbourn stated that these rotational forces set up shearing strains and stresses

between the cortex and the interior of the skull (Figure IV). Experimentally he showed that these stresses tended to be maximal in the region of the temporal and frontal poles (Figure V), where it had already been observed that contusions and lacerations were most frequent. This work has been amply substantiated by some fascinating experiments by Pudenz and Sheldon (1946), who replaced the calvaria of monkeys by transparent "Lucite" moulds which allowed high-speed cinematography of the movements of the underlying brain after impact. The sliding, rotational movement observed in the subsequent slow motion projection of the film confirmed Holbourn's hypothesis.

Mass movements of the brain may produce lesions by either the *contre coup* effect or rotational shear strains. The relative parts played by these factors are still in dispute, but it is certain that the anatomical configuration of the orbital plate and sphenoidal ridge regions predisposes to contusions and lacerations of the cortex by either mechanism.

FIGURE III.
The compression injury.

Physiological Concepts.

After the initial work by Denny-Brown and Russell, probably the next most important work was done by Walker, Kollross and Case (1944). In experimental animals these workers found an immediate electrical discharge from the cerebral cortex, followed by a pronounced increase in cortical activity — a feature which in electroencephalographic records simulates a major epileptic seizure. This activity decreases after ten to twenty seconds, and then the cortex becomes relatively inactive for a period of seconds or minutes, before it slowly begins to assume its normal activity. Evidence that the initial discharge is widespread is further suggested by the appearance of large electrical discharges in peripheral nerves and in the spinal cord.

These workers then postulated that the phenomena of concussion are due to excitation of large masses of neurons within the central nervous system, and that physiologically concussion consists of a traumatic electrical discharge from the polarized cell membranes of the central nervous system by commotion or shaking up, and that the paralytic phenomena usually associated with concussion are the normal sequelæ of physiological change. The changes are usually widespread and slowly return to normal. Although the initial changes are certainly independent of vascular effects, in the recovery stages modifications may be produced by all those conditions which normally affect the metabolic activity of the cells themselves. In human subjects it has not been possible to demonstrate the changes corresponding to the excitatory stage in animals, owing to the difficulty in obtaining early electrical records. Electroencephalographic records obtained during the following phases show a greatly diminished activity which slowly returns to normal.

Pathological Concepts.

Difficulties still beset the investigator of the pathology of intracranial trauma. Many patients still die without the finding of adequate cause at autopsy, while a great bulk of clinical material escapes post-mortem examination.

Changes in the nerve cells themselves have been studied by Courville (1946, 1945) in autopsy material, and by Groat, Windle and Magoun (1945) in experimental animals. That the changes in the nerve cells are widespread and early in their appearance is in accord with recently accepted views. But apart from the changes in the cells of the chorioid plexus which appear to indicate increased activity, there is little direct evidence to help in clinical management.

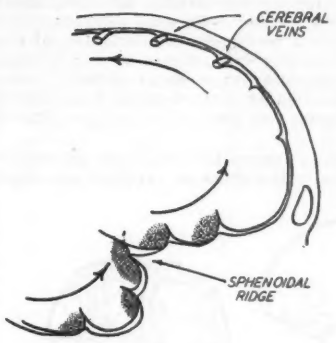

FIGURE IV.
The rotational or shearing stresses created between the brain and convolutional markings in the region of the sphenoidal ridge below and the fixed portions of the cerebral veins above.

The study of gross pathological lesions is perhaps more helpful. A number of authors have stressed the frequency of lesions in the orbital surfaces of the frontal lobes and the temporal poles (Figure VI). Whether these are due to *contre coup* mechanisms or to rotational shearing forces, the finding is of importance from the point of view of management when it is realized that after all blows on the posterior half of the head the major cerebral lesion is likely to occur in these situations. These findings also explain the fact that localizing symptoms and signs appear late in the clinical picture, because these areas are the so-called "silent areas" of the brain. The need for exploration of these areas in suspected cases is a necessary corollary of this fact.

Courville (1943) has also described hæmorrhagic lesions of the brain stem and has described clinical syndromes in acute cases, while Kremer, Russell and Smythe (1947) have described syndromes in less severe injuries when patients have survived. There seems little doubt that in this situation a complete clinical syndrome will soon be recognizable.

FIGURE V.
The vulnerable areas.

There is still little unanimity of opinion about the characteristics of traumatic cerebral œdema, and no coherent account of all the changes and their significance in relation to clinical features has appeared. Courville, in his description of the "wet brain", suggests that the essential feature of the condition is an increased production of cerebro-spinal fluid from the chorioidal plexus, while Evans and Scheinker (1943) define the condition as a state of increased perivascular and interstitial fluid which is probably due to increased permeability of the blood vessels. Greenfield (1947) suggests that a metabolic change also occurs which produces swelling, more particularly in the white matter of the brain. When the œdema is localized, this metabolic factor may be produced in the damaged area.

From the clinical point of view there are three questions which require a definite answer: (i) How is cerebral œdema to be defined and what structures does it include? (ii) Is it a local or generalized phenomenon? (iii) What part does it play in the clinical course of a head injury?

The situation is probably made clearer if the term "cerebral œdema" is applied only to swelling of the brain substance and not to increase in the quantity of cerebro-spinal fluid, even if this is shown to be an associated phenomenon. There is histological evidence to show that swelling of the brain cells occurs—cloudy swelling is observed in neurons, astrocytes and oligodendroglia, while vacuoles form in the cells of the chorioid plexus and also in the microglial cells. The axons and dendrites are also affected by patchy swelling and degenerative changes. There is an accumulation of interstitial fluid which produces a characteristic honeycombed appearance in histological sections of the affected areas. Recent workers have attributed this change to a traumatic depolarizaton of the cell membrane with resulting disturbance of the balance between the intracellular and extracellular fluids. There may be increased production of cerebro-spinal fluid, but it is probable that this factor has little effect on the clinical course of the condition.

Cerebral œdema is more frequently local than general in distribution, and naturally the areas so involved are those

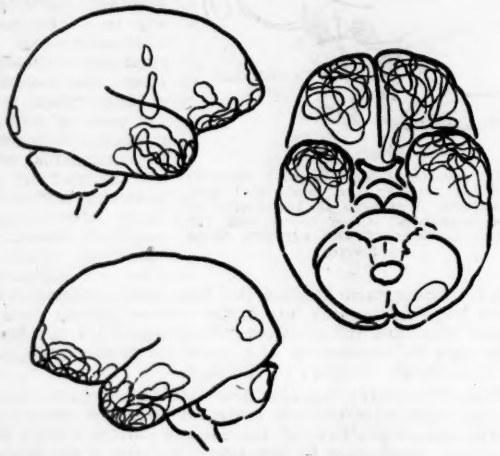

FIGURE VI.

The location of cortical lesions in a consecutive series of head injuries.

which have sustained the greatest degree of injury. They tend to occur around lacerations and contusions, and are found most frequently in the frontal and temporal regions. In these areas the convolutions may be seen to be pale and flattened, and the overlying arachnoid membrane is seen to "weep" cerebro-spinal fluid freely at operation, thus giving the typical appearance of the "wet" brain. In temporal lobe injuries the uncal region of the lobe may be herniated downwards over the free edge of the tentorium by swelling around a more superficially placed injury. Dislocation of the brain stem may be produced by the same mechanism (Figure VII). Such a localized swelling of the brain may produce a displacement of the cerebral ventricles, and is one of the conditions for which a subtemporal decompression may be indicated.

Generalized cerebral œdema is a more controversial subject, and out of a mass of conflicting thought which has surrounded this aspect of head injuries a few facts now appear to be crystallizing—namely: (i) the condition is seen when the head has been subjected to multiple injuries; (ii) it is frequent in alcoholics; (iii) it occurs when cyanosis has been present for some time.

Apart from these considerations there is little valid evidence that generalized cerebral œdema follows injury, nor is there any evidence that it is a major danger to life. In practice it would appear that many conditions previously attributed to persistent cerebral œdema (the post-contusional syndrome, prolonged confusional states *et cetera*) all have their origin in more specific processes whether pathological or psychological. For example, some cases of persistent confusion have been shown by McConnell (1944) to be due to persistent subdural effusion and have cleared up after evacuation of the fluid. The post-contusional syndrome is more often directly attributable to psychogenic and environmental factors than to organic injury.

Psychological Concepts.

From time to time papers on this subject have stressed the importance of assessing and recording the mental state of these patients in order to obtain an index of recovery or retrogression. In order to facilitate this procedure the Medical Research Council of the Privy Council published "A Glossary of Psychological Terms" (1941) to be used in cases of head injury. It has been amply shown how important and incidentally how easy it is to assess and record the state of consciousness of the patient from time to time.

Psychiatric treatment has also been introduced in the acute confusional states following head injury, and much has been learnt concerning the control of hyperactivity.

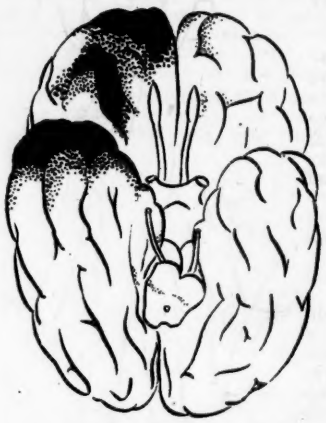

FIGURE VII.

Dislocation of the brain stem following laceration and œdema of the temporal lobe.

The assessment of organic change following a head injury is a subject which has occupied many workers in this field. Physical defects are relatively easy to assess, but intellectual and personality changes are more subtle. No series of tests has been found entirely satisfactory, mainly because of the difficulty in obtaining a level of intelligence before the accident which may be used for comparison. Further, neurotics tend to give similar responses to such tests; although Reynell's (1944) modification of the Bellevue - Weschler test has been found useful, it does seem probable, as Tooth (1947) has suggested, that the tests are measuring some factors unfavourable for recovery after head injury rather than organic impairment. In many cases a simple performance test, suggested by the nature of the patient's ordinary work and carried out in his own time at home, subsequently recorded by the patient and verified by a relative, will often give extremely useful information. At the present time far too many problem cases reach compensation boards with little more than the most superficial examination of intellectual and mental changes.

Economic Concepts.

Rehabilitation after head injuries proved a useful component in treatment of army patients, and in Great Britain these facilities were extended to some civilian patients as well, with gratifying results. Their value in peace time can be undoubted, but there are many difficulties in the organization of rehabilitation centres. The management of these patients away from their homes for long periods is difficult, while present workers' compensation and other sickness benefits only complicate the picture. The whole psychological background is subjected to powerful extrinsic influences which may make rehabilitation difficult and prolonged.

But there are many other avenues along which research and economic propaganda may be directed. The work of Cairns (1946) and of Cairns and Holbourn (1943) on the crash helmet worn by army dispatch riders demonstrated

the value of a thorough statistical survey of a group of injuries. Experimental studies on protective helmets, combined with clinical observations upon patients, showed the value of the protection afforded, and finally statistics showed how the compulsory use of the crash helmet diminished the mortality and morbidity rates of subsequent injuries in this group. Much work of a similar nature could be done from the clinical material which passes through the coroner's department.

Types of Blunt Head Injury.

The correlation of all this information is beyond the scope of this paper, which is primarily concerned with the early management of the patient with an acute head injury. But some type of classification is necessary in order to simplify the types of head injury encountered. A fundamental classification depends upon the type of impact, which may be (i) localized, (ii) generalized or (iii) intermediate in type.

The localized head injury is characterized by the fact that the maximal disturbance of the skull and brain is local and confined, thus tending to produce a limited laceration of the scalp, a depressed, comminuted or

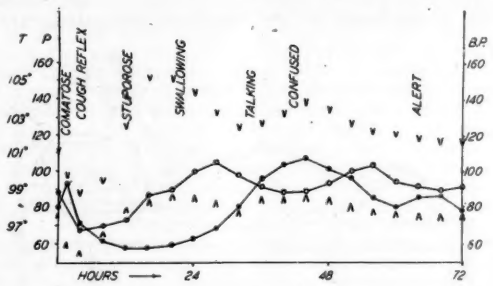

FIGURE VIII.

The four-hourly chart of a patient with moderately severe injury, who regained full consciousness within seventy-two hours; "v", systolic blood pressure; "^", diastolic blood pressure; "·—·", pulse rate; "o—o", temperature.

compound fracture, laceration of the dura and brain underlying the point of impact and a relatively slight degree of generalized brain damage. Clinical features of this type are usually limited to a short period of unconsciousness, followed by focal signs of injury, the latter depending largely on the situation and the extent of the cortical injury. There is no gross functional disturbance, and the recovery of those faculties not permanently destroyed is rapid.

In the generalized injury the disturbance of the skull and brain is widespread. In the brain, lesions may be produced by the effect of acceleration changes, by rotation within the skull and by ill-understood compression phenomena. Clinically, such an injury is characterized by a large contusion or laceration of the scalp, and most frequently by extensive fissure fractures of the skull. Within the brain itself, a widespread involvement of the nerve cells produces the phenomena of concussion. In the more severe injuries, localized contusions and lacerations of the brain occur, which later become surrounded by a zone of œdema and reaction. These may occur in areas remote from the point of injury, being most frequently seen over the frontal and temporal poles. The clinical sequence produced commences with the state of concussion and is followed by a prolonged period of unconsciousness and a slow recovery period, during which the patient passes through a definite train of abnormal psychological states. Symonds (1940) identifies the following six stages in this recovery period.

Stage I: Complete paralysis of cerebral functions, which may extend for a moment to the vital functions.

Stage II: Coma. Flaccid paralysis, with abolition of reflex function, but pulse and respiration have returned.

Stage III: Stupor. Reflex functions return; one observes movements of a simple purposive type, such as the withdrawal of a limb from a painful stimulus. The patient may respond to forcible command, but is otherwise unconscious of his surroundings.

Stage IV: Restless confusion. The patient is resistive and often violent. Voluntary movements and speech return without control or direction.

Stage V: Mental confusion. The patient's behaviour is quieter and more controlled; his speech is more coherent, but he remains disorientated.

Stage VI: Automatism. The patient responds naturally to simple questions and performs accustomed actions in an orderly way. He is still dazed and imperfectly aware of his surroundings. He repeats the same question. He has no memory of this phase in retrospect.

The use of such terms as concussion, contusion and cerebral compression as a classification of head injuries has now become inadequate. Obviously, most moderate or severe injuries will produce these pathological changes

FIGURE IX.

The "traditional" position for nursing a patient with head injury.

to a variable degree at some time or another. The transition from one stage to another is often imperceptible; furthermore, all three pathological states may be present at the one time. Thus, the clinician will experience great difficulty if he attempts to fit all cases into such a classification. Because the accepted signs of compression so often indicate established decompensation, such a practice often results in the diagnosis of a complication only after decompensation has taken place, and recovery is thereby prejudiced. When more complete correlation has been obtained between clinical features and pathological findings, it may be possible to formulate an accurate topographical and pathological classification. But until this stage is reached, a satisfactory classification of the acute blunt head injury may be based on the general or concussive effect only. This classification is thus concerned with the initial severity of the lesion, as follows:

1. Mild. Initially the patient is suffering from concussion or dazed, and this state is followed by a period of mental confusion before recovery is complete.

2. Moderate. After the initial effect of concussion has passed off, the patient still remains stuporose for a short period and then passes through the stages of confusion and automatism to full consciousness.

3. Severe. Shock usually accompanies the effects of concussion, but when this has passed off the patient remains in a state of coma or stupor for hours or days before passing through the remaining stages. Recovery is usually slow and some physical or intellectual defect is likely.

Thus the patient with a typical blunt head injury passes through phases which merge imperceptibly into one another, but the clinical state found on examination at any one time can be fitted into one of the stages described by Symonds.

The period of recovery is preceded by a relatively short period of retrograde amnesia, the duration of which is reasonably proportional to the severity of the head injury. The period of post-traumatic amnesia usually persists until the stage of recovery is reached, though it may not be complete in the later phases, when consciousness appears to be merely clouded and more dramatic and interesting events of the day are remembered.

In spite of the recent growth of knowledge it is still difficult to correlate with any degree of certainty changes in the clinical state with the pathological changes present. In the light of present knowledge it is possible to state that at the moment of impact an acceleration effect takes place within the brain, which, if of sufficient intensity, produces a widespread change in the nerve cells. The observable effects of this change are essentially paralytic in nature, and if severe enough, may produce death. In less severe injuries recovery of function takes place, first in the vital centres, and then gradually other reflexes reappear until spontaneous movements occur. The neurons will thus recover their function except in those regions where local changes—œdema, contusion, laceration or hæmorrhage—disorganize portions of the brain for longer periods, sometimes permanently. Although these areas are often "silent areas", they nevertheless play their part

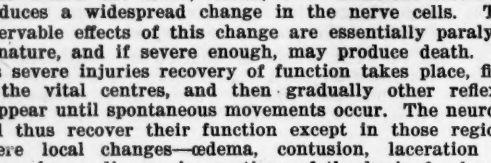

FIGURE X.
Sim's position: used when cough and swallowing reflexes are depressed.

in the normal conscious state, and the deficit, or in some cases the irritative effect of the lesion, appears to be responsible for the changes in the mental state.

Added to these changes there may be the irritative effect of the blood in the cerebro-spinal fluid, together with some increase in cerebro-spinal fluid production or diminution in its absorption. More massive extravasations of blood may occur, producing varying clinical states, which will be dealt with in another paper as complications.

In severe injuries, when progressive increase in intracranial tension occurs from extravasation of blood or cerebral swelling, the syndromes of cerebral compression eventually occur. Two clinical syndromes have been recognized by Penfield and Cone (1943), though often cases exhibit characteristics of each. The syndrome of mid-brain or tentorial compression is characterized by the following: fluctuation in the state of consciousness; dilatation of the pupil with loss of light reaction, first on one side, later on both; neck stiffness; hyperpyrexia; pyramidal signs, often paradoxical, tending to merge into a state of decerebrate rigidity.

The syndrome of medullary compression is characterized by the following: deterioration of the state of consciousness to deep coma; slowing of the pulse rate; periodic breathing; respiratory failure preceding cardiac irregularity and eventual failure.

These syndromes represent terminal conditions from which recovery is not common. In many cases progress to this state is unavoidable, but in others it is not so. Dandy (1933) has stated that without specific treatment 70% of all patients with head injuries will recovery spontaneously. Of the remainder, probably two-thirds will die in spite of all treatment. It is in the remaining 10% of the total that treatment can be improved so that the mortality may be diminished. Many of the principles applied to the surviving 70% will also be expected to diminish morbidity and economic loss.

Comment.

In the light of these clinical and pathological features it is possible to set out more specific requirements for the management of these patients. Firstly, it is necessary to observe and record the changes in the clinical state. Secondly, the natural process of recovery must be facilitated. Finally, it is necessary to prevent, anticipate and treat complications.

Observation and Recording.

Observation and recording, an essential part of the management of patients with head injuries, is concerned with the following: (i) assessment of the state of consciousness and the progressive changes in this state; (ii) recording of temperature, of pulse and respiration rates, and of the blood pressure; (iii) noting of fluid intake and output.

Little more need be said about the state of consciousness, except to emphasize the fact that these observations are by

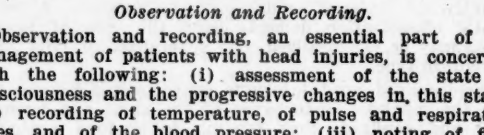

FIGURE XI.
Fowler's position: for the recovering patient.

far the most important in assessing progress or retrogression, or for obtaining an early indication of the presence of complications. The practice of recording the state of consciousness with the level of reflex activity four-hourly on a chart (Figure VIII) has considerable merit and should be introduced into all teaching hospitals.

The interpretation of the temperature, pulse and respiration records follows well-accepted principles. A caution must be added about the insidious onset of hyperpyrexia. It has become accepted that hyperthermia is always a terminal event when it follows a head injury. There is, unfortunately, far too much truth in this statement, for established, severe hyperpyrexia is usually fatal; but in many cases this state can be prevented and a life saved by employing just those principles which have saved many patients suffering from heat exhaustion in the tropics. The methods employed for controlling hyperpyrexia are simple, straightforward procedures.

1. When the temperature is found to rise above 102° F. it is recorded at half-hour intervals, and preferably *per rectum*, for in true neurogenic hyperthermia the skin temperature is no longer an accurate index of the internal body temperature.

2. When the temperature exceeds 102° F., all blankets are taken from the patient's bed and not replaced till the temperature has fallen below 100° F. Further rises in temperature are combated by cold sponging or by cold spirit sponging. In severe, persistent cases it may be necessary to sprinkle chipped ice over the patient and use an electric fan to blow cold air over the trunk.

3. Fluid administration is essential. In the established case of hyperthermia the intravenous route is used; but it must be emphasized that the use of a Rehfuss tube as

a routine measure for administration of fluid to the stuporose patient is an important prophylactic procedure.

4. Aspirin may be given in large doses, and there is some evidence that barbiturates have a remedial effect on the hyperpyrexia as well as on the hyperventilation which often accompanies this condition.

Although the clinical appearance of the tongue and of the skin is a reliable index of the state of hydration of the patient, from the practical point of view the recording of fluid intake places a responsibility on the nursing staff, and should always be insisted upon during the initial stages of treatment of a head-injury patient.

General Management.

For the most part, general management is designed to facilitate the normal processes concerned in recovery. Cerebral venous congestion and cyanosis, together with pulmonary complications, comprise the major factors operating against recovery of those patients without definite intracranial complications. The old custom of nursing a patient with a head injury in a darkened room, lying flat on his back, without a pillow to his head, is one which is dying hard amongst the nursing staffs of most hospitals. Too often a patient is seen (Figure IX) in a comatose state, without cough or swallowing reflexes, lying flat on the bed, with the tongue and jaw fallen back to obstruct the pharynx. Blood and mucus can be heard rattling up and down the trachea, while the patient is left in charge of a terrified junior nurse, who, seeing his colour become darker and darker and his respirations more and more laboured, is powerless to rebel against her established teaching. It is but the work of a few moments to turn the patient on his side and swab out his pharynx, and instantly the colour improves and the respirations become smoother and quieter. The importance of maintaining a clear airway while the comatose patient is in the lateral or Sim's position (Figure X) cannot be overestimated. When cough and swallowing reflexes return, the ideal position can best be obtained by the adoption of Fowler's position. In the past, much of the post-traumatic headache and giddiness has been due to the fact that a patient has been kept flat for long periods, and that subsequent activity has not been sufficiently graduated.

Starvation and dehydration are factors which are seen to operate adversely far too often in severe cases. Thus, in the aged, in the debilitated and in the young, particular attention must be given to nutrition and fluid balance. The widespread use of a Rehfuss tube has done much to diminish these factors, for it is then easy to administer a basal caloric requirement such as an egg flip, and sufficient fluid and salt may be given to prevent hyperthermia or urinary embarrassment.

Little more need be said about assisting the normal processes of recovery, beyond the accepted principle that the patient should be treated more like a viable human being than an inanimate, unwelcome log. This means feeding, moving, washing, and all the routine care necessary in any surgical case and so often omitted in the severe head injury case.

The routine performance of lumbar puncture is still subject to controversy. But the results obtained from this procedure are of importance in assessing prognosis, though its use as a therapeutic agent is still problematical. The following routine is advocated:

1. An initial lumbar puncture is carried out at the first examination. The fluid thus obtained sets a standard for future reference and helps in assessing prognosis.

2. Subsequent lumbar punctures are carried out if the fluid is grossly blood-stained, or if the pressure at the initial puncture exceeds 200 millimetres, and the patient survives the first thirty-six hours.

3. Lumbar puncture is performed in certain cases in which complications are expected, but not when the intracranial tension is expected to be high, making a medullary cone a possibility.

Consideration of the management in the later stages of injury is beyond the scope of this paper. It will be readily realized that, when early treatment is adequate, there will be fewer problems to deal with in the later stages of recovery from head injury. The management of the "problem case" in the earlier phases will form the basis of a later paper on the complications of head injuries.

References.

A complete list of references is given with a second paper, entitled "Complications of Closed Head Injuries", published in this issue.

COMPLICATIONS OF CLOSED HEAD INJURIES.

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THE early detection of complications which may follow the blunt head injury is possible only if a careful observation of the patient is maintained. It is in this respect that observation of changes in the following is of the utmost importance: (i) the state of consciousness; (ii) physical signs; (iii) neurological signs; (iv) temperature, pulse rate, respiration rate and blood pressure.

There can be no doubt that the best index of the progress of a patient is obtained by the frequent and careful assessment of the state of consciousness. Although minor fluctuations occur from time to time, the general trend in all patients subjected to a moderately severe head injury is a gradual transition through various stages which can be readily observed. These stages have been described in a previous paper (Hooper, 1949), and when progress is satisfactory the patient will pass through this sequence without undue prolongation of any stage. Such a sequence is represented in Figure I. The undue prolongation of any of these phases or the reversal of this normal process of recovery indicates the presence of some untoward complication (Figure II).

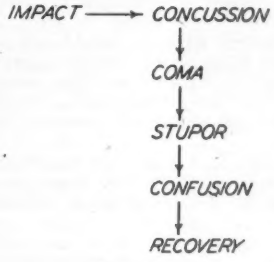

FIGURE I.

The stages in the sequence of recovery from a head injury.

The nature of this complication may be determined by the following: (i) a consideration of the time relationships in the development of the clinical picture; (ii) assessment of abnormal signs; (iii) radiographs of the skull; (iv) records of temperature, pulse rate and respirations.

For example, a retrogression of consciousness in the first forty-eight hours, together with a temporal hæmatoma, contralateral hemiparesis and a fracture through the temporal or parietal bones, will suggest an extradural hæmorrhage (Figure III), while a retrogression after twenty-four hours, without abnormal neurological signs but with a rise in both temperature and pulse rate, together with a fronto-orbital fracture, indicates a meningeal infection (Figure IV).

Abnormal physical signs are more commonly absent than present, and when they are to be observed their importance should not be under-estimated. This is particularly important when there are abnormal neurological signs.

Within certain limitations the site of injury of the scalp and the nature of the injury are of importance. Frontal contusions and lacerations will suggest fractures through the frontal sinuses with subsequent risk of meningitis. Temporal hæmatomata may suggest extradural hæmorrhage in this situation, while in the occipital region a superficial injury may give the direction of the blow, for in this situation they are frequently associated with frontal lobe contusions. Less frequently they may be associated with a fracture-dislocation of the cervical part

of the spine. Hæmorrhage from the nose or ear, especially when there is associated leakage of cerebro-spinal fluid, always suggests fracture of the base of the skull, a type of lesion indicating a severe injury and carrying with it the additional risk of infection.

Neck stiffness is a sign of the utmost importance. In most cases it serves to indicate the presence of a subarachnoid hæmorrhage, but it may indicate meningeal infection or injury to the cervical part of the spine.

When one is assessing the importance of abnormal neurological signs it is necessary to ascertain the level of reflex activity in comatose and stuporose patients. The observation of reflex activity may be the only reliable guide for assessing the progress of a patient examined in

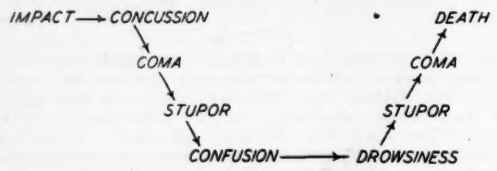

FIGURE II.

The sequence of stages of recovery and retrogression which indicates the presence of a complication.

the early stages after a head injury. Thus it is very important when describing the comatose patient to refer to the level of reflex activity. Corneal reflexes, swallowing reflex, cough reflex, the presence or absence of deep reflexes and the reflex response to pain should be noted.

The pupillary reaction requires detailed consideration. In traumatic lesions two main types of disturbance occur: (i) the dilated pupil, which is inactive to light when either eye is illuminated; (ii) the dilated pupil, which is inactive to light in the same eye, but reacts consensually to light in the opposite eye.

The former is primarily due to a lesion of the third nerve, which may be caused by a direct and immediate injury to the nerve itself or by a more gradual process

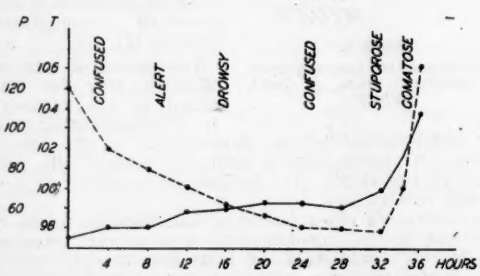

FIGURE III.

The chart of a patient who developed a temporal hæmatoma and had sustained a fracture in the temporal region after a fronto-temporal impact; "·——·", pulse rate; "·—·", temperature.

in which there is the slow development of a fixed, dilated pupil followed by fixation and dilatation of the pupil on the opposite side. The exact mechanism of this process is not known with certainty, but in many instances the site of the lesion lies in that portion of the nerve which passes along the free edge of the tentorium and becomes pressed upon by the uncinate portion of the temporal lobe when a tentorial herniation is present. Often this explanation is incomplete, for the pupillary change is seen on the side opposite the lesion in about 30% of the cases.

In the second instance the oculomotor portion of the reflex pathway is intact, but there is a lesion of the optic nerve or chiasm. It is this lesion which often causes difficulty if the consensual light reaction is forgotten.

Pyramidal tract signs can be adequately interpreted only in relation to other factors, but a few generalizations are possible. A cerebral contusion rarely produces more than an incomplete paresis, which soon shows signs of recovery. Weakness of the face and hand without involvement of the leg is the usual sequence when pyramidal signs are present in extradural hæmorrhages.

Sensory defects are uncommon and when demonstrable indicate a parietal lesion of some severity.

Evaluation of the Temperature, Pulse and Respiration Records.

The importance of the temperature, pulse and respiration records can be assessed only when they are considered with other clinical findings and when the time relation of the various changes is observed.

Pyrexia is of significance in the early detection of complications. Temperatures up to 101° F. may often be due to a subarachnoid hæmorrhage, and if blood is present in moderate amount in the cerebro-spinal fluid we must be satisfied with this diagnosis. For levels higher than 101° F. this explanation must be considered insufficient and a more diligent search for a cause must be made.

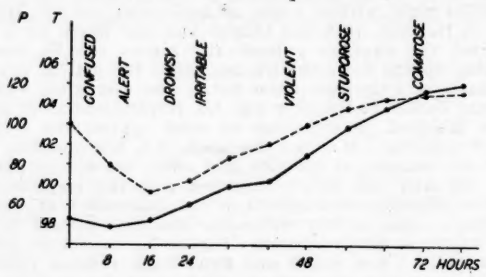

FIGURE IV.

The chart of a patient who became comatose seventy-two hours after a frontal injury; "·——·", pulse rate; "·—·", temperature.

The following must then be considered: meningitis, bronchopneumonia, urinary infections (especially in the female), starvation and dehydration, hyperthermia (neurogenic).

Variations in the pulse rate and blood pressure are, by themselves, often unreliable, but taken in conjunction with the rest of the clinical picture they may be of considerable importance. In the early stages after injury a weak, rapid pulse suggests surgical shock, and this condition must be given due attention. It must be remembered that prompt resuscitative measures produce favourable results in head injuries more rapidly than in equivalent limb injuries, and the persistence of a rapid pulse rate may indicate the presence of extracranial complications. In the later stages a rise in pulse rate will serve to indicate the conditions which have been mentioned under pyrexia. It is the slowing pulse rate and rising blood pressure which constitute the well-known signs of cerebral compression. Although often present, the duration of this stage may be short, and it may soon be overshadowed by other changes associated with fever or circulatory failure or following excitation.

The observation of changes in respiration is important, but again it is frequently found that undue importance is attributed to these changes: for example, the appearance of Cheyne-Stokes respiration is taken as an irreversible change and a precursor of death. This is not so in all cases, and its presence is an indication to consider what can be done for the patient rather than as an excuse for the cessation of all treatment.

Amongst blunt head injuries large extravasations of blood produce by far the greatest proportion of complications in the acute stages, and a still considerable proportion in the later stages. For the purpose of this paper the discussion will be confined to these complications.

Subarachnoid Hæmorrhage.

Subarachnoid hæmorrhage is the common counterpart of most head injuries, and it is perhaps wrong to include it in a discussion of complications; but it is often the cause of early deterioration of consciousness after a simple fall. In children particularly its occurrence may cause alarm. Its presence is suggested by mild fever, neck stiffness and irritability; drowsiness and confusion occur less frequently in adults, though children may pass into a stuporose state after several hours. Generalized convulsions are not uncommon manifestations in both children and adults. Confirmation is obtained by lumbar puncture, and this procedure is the only means of distinguishing it from meningitis. Massive types of subarachnoid hæmorrhage usually arise from the tearing of vessels at the base of the brain in the region of the frontal lobes or the Sylvian fissure, and are usually fatal in the early stages.

Intracerebral Hæmorrhage.

Intracerebral hæmorrhage is uncommon, but two distinct forms may be seen from time to time.

1. The early form is seen more commonly with severe injuries, but does occur with injuries of varying degree. It is manifested by an early flaccid hemiplegia, and it may be associated with subdural and subarachnoid extravasations. In about 75% of the fatal cases rupture occurs into the ventricles or subarachnoid spaces. In the non-fatal cases the clinical picture is one of an expanding intracerebral lesion which as such becomes an indication for exploration.

2. The delayed form, or *"traumatische Spätapoplexie"*, was first described by Bollinger in 1891. The exact pathology of this condition still remains uncertain. This complication may follow more or less trivial injuries, not necessarily associated with loss of consciousness in the initial stage. The delayed hæmorrhage may occur days or even months after the injury. It has been suggested that the condition results from a fresh hæmorrhage into the cavity of a chronic intracerebral hæmatoma, but it is difficult to fit all the recorded cases into the group. The condition should always be considered when there are signs of rapid deterioration of consciousness with hemiplegia days or even weeks after an injury. Occasionally epileptic seizures occur. The diagnosis of the position of the hæmorrhage is usually made from the clinical signs, but sometimes a ventriculogram is necessary before the hæmatoma can be located with the exploring needle. In many cases the clot can be removed by aspiration, but in the intermediate stage before liquefaction has taken place the clot may be tough and gelatinous, and it may be necessary to raise an osteoplastic flap and evacuate the clot after incising the overlying cortex. The latter is always stained in colour, giving the clue to the position of the clot.

Subdural Hæmorrhage.

Hæmorrhage into the subdural space is probably the commonest complication of head injury, and it certainly represents the most important operable lesion.

The traumatic subdural hæmatoma may occur in all situations, and though it is most frequently seen in the parietal region it is not infrequently found in the temporal region and may even occur in the posterior fossa. These situations are not infrequent in infants.

Recent experiments with the "Lucite" calvarium described in the previous paper (Hooper, 1949) show that it is possible to produce the typical parasagittal subdural hæmorrhage in animals by those mechanisms which had been thought to operate in humans. It was also shown that those factors which were considered to predispose to the formation of the hæmatoma, for example, dehydration and direction of the impact—operated in the same way in the experimental animal.

Although the origin of the hæmorrhage is not often determined either at operation or at autopsy, it is assumed that the hæmorrhage is most frequently venous in origin, and that it originates from the tearing of the veins passing from the brain across the subdural space to enter the sagittal sinus above or below the sphenoparietal sinus in the region of the Sylvian fissure. In infants the great vein of Galen or other veins passing to the tentorium may be torn. But in some cases the hæmorrhage originates from a laceration of the cortex, when both small arteries and veins contribute. Here also there may be some escape of cerebro-spinal fluid into the blood, with subsequent dilution, till all stages may be obtained between a blood-stained effusion of cerebro-spinal fluid and solid blood clot.

The subsequent changes in the hæmatoma appear to be well recognized. The presence of a blood clot causes the formation of a membrane of granulation tissue, first on the inner surface of the dura and then over the inner surface of the clot as well. Meanwhile, inside the clot the complex molecules are gradually broken down to more numerous, smaller groups with a subsequent rise in the osmotic pressure of the fluid contained in the subdural space. Until the molecules are small enough to pass through the membranes already formed, this rise in osmotic pressure is followed by the absorption of fluid into the subdural collection, which now acts as an expanding intracranial lesion, producing all the phenomena associated with such a condition.

In direct relation to this sequence it is possible to describe three clinical types: (i) the acute form, (ii) the delayed or chronic progressive form, and (iii) the chronic or the chronic persistent form.

Acute Subdural Hæmorrhage.

Acute subdural hæmorrhage is usually associated with lacerations of the cortex and is thus found to a greater or lesser degree in severe injuries of the cortex. The extravasated blood may come from both veins and arteries, so that there is a relatively rapid accumulation of blood clot with the early production of signs. This development may occur before the patient has had time to recover from the concussive effect on the brain, and in such cases the steady deterioration effectively masks the presence of any localized or recoverable lesion.

In less severe cases the hæmorrhage occurs more slowly and partial recovery from the immediate effects of the injury occurs before the accumulation of blood produces any effect. Later the patient becomes confused and stuporose, though from time to time a certain amount of compensation may occur, producing transient remissions manifest by a return of awareness for short periods. After an interval of one to five days from the initial injury coma deepens and the signs of a compression syndrome become unmistakable. Although localizing signs are uncommon in this group, especially with hæmorrhages over the convexity of the hemisphere, the localized subdural collection in the region of the sphenoidal ridge may produce the typical sequence of facial and upper limb weakness on the contralateral side, with dilatation and inactive light reflex in the ipsilateral eye.

Delayed Subdural Hæmorrhage.

Delayed subdural hæmorrhage is the most familiar form and is generally termed "chronic subdural hæmatoma". But this is not sufficiently descriptive unless the term "chronic progressive hæmatoma" is used.

The onset of symptoms corresponds to the expansion of the subdural collection as a result of the osmotic effect. The syndromes produced are varied in form, but the following sequence may be taken as typical.

The hæmorrhage is produced by an injury of moderate or mild degree, from the initial effects of which recovery takes place. Then, for a variable period, headache, drowsiness, impaired concentration and memory, and occasionally actual weakness of the limbs may be present. The pupils are often sluggish and unequal and a low-grade papilœdema may be present. It is characteristic that these symptoms vary in degree from time to time. As the accumulation of fluid increases and compensation fails, drowsiness passes into stupor and coma. Gross pyramidal signs may be present. The pupils fail to react to light and eventually signs of mid-brain or medullary compression occur.

Chronic Subdural Hæmorrhage.

When complete breakdown of the protein molecules has occurred before any decompensation has taken place, the effusion may resolve as the fluid becomes reabsorbed and the lining membranes also disappear. But in some cases reabsorption is imperfect, and thus the chronic form remains. Perhaps "chronic persistent" form is the better term. The continuance of the condition may be due to a number of factors, as the following:

1. Small hæmorrhages may occur from the vascular granulation tissue which forms the wall of the resolving hæmatoma.

2. Atrophic shrinkage may occur in the underlying brain during the period of development of the hæmatoma, thus hindering the reexpansion of the brain and the obliteration of the subdural space.

3. In infants the presence of the hæmatoma causes cerebral atrophy on the one hand and expansion of the skull on the other. Even after evacuation of the subdural fluid in these infants re-collection is frequent because the brain cannot expand to fill the space between the atrophic brain and the hydrocephalic skull.

In adults this form is characterized by headaches and mild intellectual defects, while in children retardation of development is common.

The Subdural Effusion.

Some mention must be made at this stage of the subdural effusion or subdural hygroma. This condition is produced by the leakage of cerebro-spinal fluid from the subarachnoid spaces into the subdural space, from which it is not readily absorbed. Two forms of this condition may be distinguished:

1. The acute subdural hygroma. This is presumed to be due to the passage of cerebro-spinal fluid into the subdural space through a valvular tear in the arachnoid membrane. Fluid accumulates under tension in the subdural space and produces all the signs of a rapidly increasing intracranial lesion. When the dura is incised at operation, clear or slightly blood-stained cerebro-spinal fluid spurts out through the opening under considerable tension and in considerable amount. Subsequent improvement may be dramatic.

2. The chronic form. This may be produced in the same manner as the acute form, while on the other hand it may represent an effusion through a serous membrane—the arachnoid membrane—in the region of an injury. Such a process is often observed when a cortical laceration is exposed at operation. This form must also include the late forms of subdural hæmatomata. In such cases the accumulation of fluid hinders cerebro-spinal fluid absorption and in general all those reparative processes in the brain which eventually lead to recovery. Hence, in these cases we find undue prolongation of the period of unconsciousness and delayed recovery of intellectual function. Removal of the fluid by craniotomy, although not always dramatic, hastens the rate of subsequent recovery.

The Extradural Hæmorrhage.

The extradural hæmorrhage may arise from a number of different sources. Although it is true that it most frequently follows tearing of the middle meningeal vessels, the hæmorrhage may originate from diploic channels or from torn dural sinuses. Whatever its origin may be, it is still most commonly found in the temporal region. Nevertheless it must be realized that other situations may be involved and it must be thought of when, in the skull radiographs, a fracture line crosses a vascular marking.

The mortality associated with extradural hæmorrhages is very high, and in order to justify the principle that the suspicion of an extradural hæmatoma is sufficient indication for exploration, the following figures, published by Munro and Maltby, are given.

1. The incidence varies between 5% and 10% of patients admitted to general hospitals.

2. A lucid interval occurs in only 40% of cases; the duration may be as short as one and a half hours and as long as sixteen days, the average duration being twenty-eight hours.

3. Pyramidal signs, often ipsilateral, occur in about 50% of cases.

4. Of patients first examined in coma, 70% died.

5. Of the fatal cases, in 90% frankly blood-stained cerebro-spinal fluid was present; but so it was in 60% of the recoveries.

6. There was a case mortality rate of 69% and an operative mortality rate of 50%.

Thus it would appear that there are two main factors upon which the prognosis depends: (i) the severity of the injury—prognosis is poor when coma is present from the beginning or develops early; (ii) the state of consciousness when operative intervention is undertaken.

Over the first factor we have no control, but too frequently patients are allowed to develop the complete clinical picture; even the necessary delay in setting up the operating theatre allows such a patient to lapse into coma, so that the prognosis becomes poor. The syndrome of the established extradural hæmorrhage is well known, and it is necessary only to underline or accentuate certain points. (i) A lucid interval may not be present. (ii) The patient may not be stuporose or even drowsy. (iii) Œdema of the temporal fossa or of the orbit on the affected side is an important sign. (iv) The suspicion of an extradural hæmatoma is an absolute indication for an immediate radiograph of the skull. (v) the suspicion of an extradural hæmorrhage combined with X-ray evidence of a fracture line crossing a vascular channel in the bone is sufficient indication for operation, even when the clinical signs are minimal and the patient is still conscious.

Only by the adoption of such a rigorous routine will the high mortality of this condition be reduced.

Cerebral Œdema.

The subject of cerebral œdema must be included in any discussion on complications. Naturally, it is difficult to say in what manner cerebral œdema affects the clinical manifestations of a head injury, and when it can really be said to be a complication in the common meaning of the term. For the most part there is little valid evidence that cerebral œdema produces any characteristic clinical features, and the diagnosis is rarely based on more suggestive information than a raised cerebro-spinal fluid pressure, headache and delayed recovery. In spite of this difficulty it is important to know what factors increase the production of cerebral œdema and what treatment is necessary to limit its effects.

Some elaboration of the part played by cerebral œdema is possible. It will be readily understood that the presence of œdema of greater or lesser degree around a focus of cerebral injury influences the time necessary for its resolution. In certain situations, notably in the temporal lobe, the presence of a considerable amount of œdema leads to distortion of the brain, which may produce a further increase in œdema—a process which may continue till irreversible changes take place. It is in such conditions that a local decompression operation may be of considerable help. The generalized formation of œdema is the probable cause of delayed recovery of those subjected to multiple injuries, of alcoholics, and of those patients whose airway has been obstructed for long periods or in whom bronchopneumonia has developed.

In considering what treatment is necessary to limit the effects of cerebral œdema, some caution is necessary. Intracranial tension may be diminished in many ways, and it is necessary to restrict the methods used to those which do not produce untoward secondary effects either inside the cranium or within the body generally. For example, in the presence of a small recent subdural hæmorrhage, the sudden lowering of the intracranial pressure may initiate a further access of hæmorrhage. It is hardly necessary to point out that dehydration of a patient with bronchopneumonia, fever and some degree of exhaustion is bad treatment.

It is not sufficiently realized that there are other methods by which the intracranial pressure can be reduced, and

of these the adoption of the sitting posture and free unrestricted respiratory movements are most important.

To sum up, it seems reasonable to assume that localized œdema is a frequent sequel of severe injury. It rarely requires more specific treatment than that normally regarded as necessary for the associated contusion or laceration. It must be conceded that generalized cerebral œdema occurs under some conditions. In such cases additional dehydrating measures may be instituted if the patient's general condition is satisfactory and there are no intracranial hæmorrhages to contraindicate such procedures. Temporal decompression should be limited to those cases in which œdema has been demonstrated by exploratory trephining or by displacement of the ventricles shown in air studies of the brain.

Treatment.

The management of head injuries may be broadly divided into three separate functions: observation, prevention of complications, surgical intervention.

FIGURE V.

The situation of the incision through which an exploratory trephine or burr hole is made. The dotted line indicates the area of infiltration with local anæsthetic.

The observation and recording of progress have been outlined in a previous paper (Hooper, 1949) and no further comments will be made on the prevention of extracranial complications which account for many of the deaths of patients who survive the first few days after injury. With adequate management along these lines about 70% of all patients with head injuries will recover without any active treatment. The remaining 30% represent those patients whose condition deteriorates gradually, intermittently or even catastrophically. These patients may be divided into the following groups: (i) those destined for an irrevocably early death as a result of gross brain damage; (ii) those who exhibit the signs and symptoms of a typical intracranial hæmorrhage; no difficulty will be experienced in making the decision to operate on these patients, nor will there be much diversity of opinion on the operative procedure to be adopted; (iii) a considerable proportion, for whom a definite pre-operative diagnosis is not possible in spite of adequate examination and observation, together with the results of lumbar puncture and radiographs of the skull.

It is in this last group, when progress may be just "unsatisfactory", that some plan of procedure appears to be necessary. The following may be taken as indications for operative intervention in such "indefinite" cases: (i) increasing depth of unconsciousness in a patient who has survived for eighteen to twenty-four hours; (ii) the appearance of focal signs of increasing severity; (iii) failure to improve after forty-eight hours of conservative treatment.

These indications are mainly applicable to patients in the early or acute stage of their injury. The initial waiting period before operation is undertaken must be somewhat variable, for the interval may be reduced when there is evidence that the initial injury was not severe; while in patients who have had a severe injury early intervention is usually unprofitable and sometimes definitely unwise.

The procedure advocated, once the decision to operate is made, is as follows.

1. The head is shaved and atropine is given. An intravenous drip infusion is set up and the patient is placed on the operating table. The areas for operation are infiltrated with 2% procaine hydrochloride solution containing a few minims of 1 in 1000 adrenaline solution.

2. A small craniotomy opening about two centimetres in diameter is made in the parieto-temporal region on each side (Figure V). Subsequent procedures will depend upon what is found at this stage.

3. If an extradural clot is found the incision is extended downwards to the level of the zygomatic arch (Figure VI). The temporal muscle is widely retracted and a routine

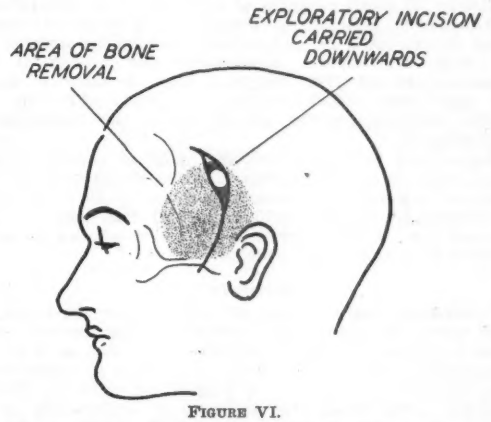

FIGURE VI.

The conversion of the exploratory incision into a subtemporal decompression.

subtemporal decompression is carried out by nibbling the bone away in a downwards direction. The subsequent bone defect should be at least five centimetres in diameter. The extradural clot is removed and the bleeding vessel is located. It is then closed by a suture, a silver clip, wax or diathermy, according to the situation. In early cases the dura is then opened and the subdural space and underlying cortex are inspected. Hæmostasis is completed and the wound is closed in the manner of a routine subtemporal decompression.

4. If a subdural clot is present the depth of the clot at the level of the initial bone opening is estimated by a brain needle or a blunt probe. If the probe passes up towards the vertex, the clot probably originates in the region of the sagittal sinus. When this is the case a further bone opening should be made more medially and posteriorly in the parietal region. From these two openings an attempt is made to wash or suck out the clot. Sometimes this procedure is inadequate and a parietal osteoplastic flap is necessary for adequate removal of the clot. When this has been necessary great care must be taken in subsequent hæmostasis, and after the bone flap is replaced it should not be fixed too rigidly, but allowed to ride if a subsequent increase in intracranial pressure occurs. Drainage tubes may be inserted into the subdural space and used for the instillation of penicillin. Not only does this diminish the possibility of infection, but the penicillin is readily absorbed and may have the added virtue of diminishing the coagulability of the blood in the subdural space.

If the probe passes downwards, hæmorrhage probably originates from the region of the temporal pole. The incision should then be continued downwards and a formal subtemporal decompression completed. The clot is cleared away and any grossly lacerated portions of the brain are gently removed by suction, while the cortical vessels are sealed by the application of the diathermy or silver clips. Complete hæmostasis is essential, and after this has been obtained the wound is closed as before. Again, it is wise to insert a drain into the subdural space.

5. If a definite cortical contusion is seen, it is wise to carry the incision down into the temporal fossa and complete a formal subtemporal decompression.

6. If no localized pathological lesion is seen, a brain needle is gently inserted into the brain in various directions and, if possible, it should be introduced into the lateral ventricle and an estimate of the intraventricular pressure made. If the brain looks generally moist and swollen, with pale flat convolutions, and if the intraventricular pressure is high, it is wise to complete a routine decompression. If neither of these conditions is present this is probably unnecessary.

7. If doubt still exists concerning the presence of a localized lesion, air should be introduced into the lateral ventricle. A small quantity of air (five to ten millilitres) will suffice to indicate the presence of a unilateral lesion when antero-posterior and postero-anterior radiographs of the skull are taken. Lacerations and œdema of the under-surface of the temporal lobe produce a small but appreciable shift of the ventricles, while in the larger hæmorrhages the asymmetry of the ventricles is unmistakable.

Subsequent surgical procedures will, of course, depend upon these findings. However, if no disturbance of the ventricular system can be seen there can be no intracranial contraindication for dehydration, and this may now be carried out, though careful observation of the patient's general condition must continue.

Conclusion.

It must be realized from the outset that the mortality and morbidity which now prevail among patients who have sustained a head injury can be reduced only by the anticipation of trouble. Once a syndrome of compression has become established, the chances of recovery decline rapidly. But when the principle of exploration in all doubtful cases is adhered to there will still be deaths for which neither the surgeon nor the pathologist will be able to find an adequate explanation. The need for more detailed clinical records and for comparative studies of the formalin-hardened brain *post mortem* is all too apparent.

Acknowledgements.

Acknowledgements must be made to the late W. A. Hailes, F.R.C.S., and to A. E. Brown, M.B., Ch.B., F.R.A.C.S., for their help in the compilation of this and the preceding paper (Hooper, 1949), and to Mr. R. Evans for the drawings from which Figures I to III in this paper were reproduced.

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[1] These references apply both to this paper and to the previous paper (Hooper, 1949).

Reports of Cases.

A CASE OF HIDRADENOMA OF THE VULVAL REGION.

By BRUCE BOSCENCE, M.B., M.S., F.R.C.S.E., F.R.A.C.S.,
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CASES of sweat gland tumours of the vulva (hidradenoma) are of great interest and sufficiently rare to justify a brief report.

Clinical Record.

G.N., a married woman, aged forty years, was first examined in February, 1948, when she complained of a sore in the perineum which had been present for six months, which was increasing in size, and which bled when irritated by her clothing. Investigation of her family and personal history revealed nothing of importance.

On examination of the patient, a firm, rounded, dark tumour was present in the perineum adjacent to the posterior end of the left *labium majus*. It was about 1·5 centimetres in diameter, and although the surrounding tissues were slightly indurated, it was movable and not fixed to the deeper structures. The surface was ulcerated and bled easily. The inguinal lymph glands on both sides were slightly enlarged.

No other abnormality was found. A diagnosis of epithelioma was made, and the tumour was widely removed together with the inguinal glands.

The patient's recovery was uneventful, and she has remained well since.

Biopsy Report.

The description of the specimen is as follows. The tumour from the labial region is dark in colour and measures 1·5 centimetres in diameter. In its deeper part, it is closely attached to subcutaneous tissues, but on the surface it herniates through the skin.

Microscopically, the tumour has the structure of an adenoma (Figure I). The ducts are both small and large and are lined by a single layer of cuboidal cells with a deeply-staining nucleus. The cells are non-ciliated and no

mitotic figures were found. The surrounding tissue is compressed to form a pseudocapsule. No metastases were present in the lymph glands.

Discussion.

Sweat glands vary in structure between the acinous sebaceous glands secreting sebaceous material and the tubular sweat glands producing watery sweat.

Maximow classifies these glands into three main groups: (i) merocrine, in which the epithelium remains intact during secretion; (ii) aprocrine, in which portions of the cell cytoplasm are extruded to form the secretion (these glands are most commonly found in the axillæ, labia, perineum, groins and *mons pubis*); (iii) holocrine, in which the epithelial cells degenerate completely during secretion (sebaceous and mammary glands belong to this group).

Hidradenoma of the vulva may arise from merocrine glands, but McDonald (1941) has suggested that the origin is more probably from the apocrine glands.

FIGURE I. Hidradenoma (× 60).

A detailed description of this condition, is given by Novak (1947) and by Novak and Stevenson (1946). In the latter paper, 16 cases are described, the youngest patient being aged twenty-eight years and the oldest sixty-seven years. The commonest site is the *labium majus*, and the next commonest sites are the paralabial skin and perineum. The tumours are seldom larger than a cherry, and may be firm like a fibroma, but often the surface is soft with a tendency to hæmorrhage. The extrusion of reddish, pulpy material is a reliable diagnostic sign. As a rule these tumours cause the patient little inconvenience, and are benign. Occasionally, adenocarcinomatous change has taken place. When incompletely removed they may recur locally. If the condition is kept in mind, the correct pre-operative diagnosis can usually be made. Histologically the tumour may sometimes be mistaken for adenocarcinoma.

Comment.

In this case an incorrect pre-operative diagnosis was made, and the operation became more extensive than was necessary. Had a frozen section been prepared at operation, the true nature of the tumour would have been revealed.

Summary.

1. A case of benign sweat gland tumour of the vulval region (hidradenoma) is presented.

2. The signs, symptoms and pathology are briefly discussed.

3. Attention is drawn to the desirability of making the correct diagnosis in these cases so that satisfactory treatment can be given without an extensive operation.

Acknowledgement.

I wish to thank Dr. C. Duncan, of the Commonwealth Health Department, for the biopsy report and the photomicrograph.

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Reviews.

A HANDBOOK OF SURGERY.

PART IV of the eighth edition of "A Short Practice of Surgery" by Bailey and Love has now been published.[1] The part deals with such important surgical matters as the head, spine, nerves, breast, thorax, larynx and infections of the hand. It may be said at once that as far as it is possible in a book which is avowedly a summary, the teaching is excellent. As usual, the skilful use of different types, spacing and diagrams has done much to clarify difficult sections. A very good example of this is the diagrammatic representation of the pathology of the breast on page 748. The only criticism to be made is that in the section on radiotherapy to mammary carcinoma no mention is made of the use of radon needles.

PART V of "A Short Practice of Surgery" by Bailey and Love has now appeared.[2] This part deals with bones, joints, muscles, tendons and deformities. Under the heading of deformities is included a great deal that is usually placed under the heading "orthopædics". The most notable thing about the section is the masterly way in which the great mass of material is handled. Although much use is made as always of photographs, paragraphing *et cetera*, a great deal of the success achieved is due to the authors' command of language. They have mastered the difficult art of clear writing. So good indeed is this part, that even an advanced surgeon will gain much help from the skilful method of summarizing adopted by the authors.

JOINT MANIPULATION.

THE production of a second edition of "Joint Manipulation, Volume I, The Extremities", by James Mennell, ten years after the first edition was published, is most unusual, in view of the fact that the first edition, Volume II, "The Spinal Column", has not yet appeared.[3] Three new and two alternative techniques in joint investigation have been added. Two concern the manipulation of the shoulder joint and one concerns manipulation of the joints of the cuboid. Some new illustrations and X-ray photographs are included.

[1] "A Short Practice of Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., and R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.A.C.S., F.I.C.S.; Eighth Edition; Part Four; 1949. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 196, with illustrations, some of them coloured. Price: £2 12s. 6d. the set. Not sold separately.

[2] "A Short Practice of Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., and R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.A.C.S., F.I.C.S.; Eighth Edition; Part V; 1949. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 252, with 202 illustrations, of which 11 are coloured. Price: £2 12s. 6d. the set. No sold separately.

[3] "The Science and Art of Joint Manipulation", by James Mennell, M.A., M.D., B.C. (Cantab.); Second Edition; Volume I; 1949. London: J. and A. Churchill, Limited. 9¼" × 7", pp. 236, with 299 illustrations. Price: 24s.

The use of the word "tension" in the first edition has been replaced by the more suitable term "traction".

The author postulates that symptoms of discomfort, weakness and even pain with widespread radiation may be due to a "joint lesion". A "joint lesion" is defined as "some disordered function within a joint and therefore of necessity there is some abnormality either in the anatomical relationship between the joint surfaces or some lack of normal physiological movement between them, or more often both". The "joint lesion" is attributed to ligamentous strain causing the articular surfaces to "seize up", or to minor subluxation not detected in X-ray examination or to loss of elasticity in ligaments.

The statement that pain referred from a "joint lesion" is transmitted via sympathetic nerve fibres is without any foundation. A "joint lesion" is diagnosed by careful examination of joint movements whether or no they are under voluntary control. The technique for examination and for treatment is identical, except that examination omits the administration of the final impressed force, which is calculated to aid the restriction of normality with regard to any function that is found to be imperfect.

No attempt is made to discuss the diagnosis of diseases or injuries affecting joints. The techniques for manipulation of joints of the extremities are sound. The methods are the same whether the joint is the site of adhesions, of arthritis or of a hypothetical "joint lesion". The importance of movements which are not under voluntary control is correctly stressed. These movements are investigated before the movements of the joint which are under voluntary control are determined or secured by manipulation.

We agree with the author's request that where symptoms do not fit in with routine diagnosis or do not respond to treatment, the nerve supply to the part should be scrutinized for the possibility of referred pain and the joints examined in the neighbourhood of the origin of or near the path of the nerve found to be involved. Until our knowledge of the exact movements of joints has been improved and until we learn more about the origin and transmission of pain arising in or around joints, treatment by manipulation must be mainly empirical.

CARDIO-VASCULAR DISEASE.

A COMPREHENSIVE survey of physiological, anatomical, pathological and clinical aspects of the subject has been the object of Louis H. Sigler in "Cardiovascular Disease: Fundamentals, Differential Diagnosis, Prognosis and Treatment".[1] However, in a volume of 551 pages it is manifestly impossible to describe adequately details of anatomy and physiology; this portion of the text is relatively unsatisfying owing to its factual nature and style, although the author has presented the subjects in the light of their importance in physical examination of the patient and in the assessment of disease of the cardio-vascular system.

Clinical methods and physical signs are given in detail as far as these concern the system, and the opening chapter on the incidence of cardio-vascular disease is a valuable review.

The chapters dealing with disease of the heart and blood vessels, coloured as they are by the author's personal experience, are not only of greater interest, but also are written more attractively and provide a stimulus to further inquiry into the problems of pathogenesis, diagnosis and treatment. The importance of psychogenic factors in simulating cardio-vascular disease and in the management of disease is adequately stressed, and one chapter is devoted to "psychosomatic cardiovascular abnormalities".

It is regrettable that illustrative electrocardiograms are not included, reference being made to appropriate sections of the author's book on the electrocardiogram. For each chapter an extensive list of references is given, but it is noted that there are relatively very few directing attention to work of the English school of cardiologists.

The compact volume covers the whole field of affections of the cardio-vascular system, including shock, and can be recommended particularly for use by the general practitioner who desires to understand not only the clinical manifestations and treatment of the diseases, but also the fundamental processes affecting the individual as a whole in health and in disease.

[1] "Cardiovascular Disease: Fundamentals, Differential Diagnosis, Prognosis and Treatment", by Louis H. Sigler, M.D., F.A.C.P.; 1949. New York: Grune and Stratton, Incorporated. 9" x 6", pp. 574, with many illustrations. Price: $10.00.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Early Carcinoma of the Uterine Cervix: Pathogenesis and Detection"; revised and augmented edition by Hansjakob Wespi, M.D., translation by Marie Schiller, Ph.D., foreword and survey by Walter Schiller, M.D.; 1949. New York: Grune and Stratton, Incorporated. 8¾" x 5¼", pp. 282. Price: $6.50.

Deals mainly with the early diagnosis and the development of carcinoma of the cervix.

"An Epitome of the Laboratory Diagnosis and Treatment of Tropical Diseases", by Horace M. Shelley, F.R.F.P.S., M.R.C.P. (London), D.T.M. and H. (England); Second Edition; 1949. London: Staples Press, Limited. New York: Staples Press, Incorporated. 8¾" x 5½", pp. 150, with few illustrations. Price: 25s.

Written for the medical practitioner in the tropics to supply him with the essential details of laboratory diagnosis and treatment of tropical diseases.

"Industrial Hygiene and Toxicology", by Frank A. Patty; Volume II; 1949. New York: Interscience Publishers, Incorporated. 9¾" x 6½", pp. 600. Price: $15.00.

Supplementary to Volume I, a review of which was published in May, 1949.

"Medical Clinics on Bone Diseases: A Text and Atlas", by I. Snapper, M.D.; Second Completely Revised and Augmented Edition; 1949. New York: Interscience Publishers, Incorporated. London: Interscience Publishers, Limited. 10¾" x 8¼", pp. 324, with many illustrations. Price: $20.00.

Includes observations and X-ray pictures specially chosen to elucidate the problems of bone diseases.

"Biomicroscopy of the Eye: Slit Lamp Microscopy of the Living Eye", by M. L. Berliner, M.D.; Volume II; 1949. New York: Paul B. Hoeber, Incorporated. 10" x 6¾", pp. 831, with 202 illustrations. Price: two-volume set $50.00, Volume II alone $35.00.

Volume I was published in 1943; it has been reprinted with some corrections and additions.

"The 1949 Year Book of Medicine (July, 1948, to May, 1949)", edited by Paul B. Beeson, M.D., J. Burns Amberson, M.D., George R. Minot, M.D., S.D., F.R.C.P. (Edinburgh and London), William B. Castle, M.D., S.M. (Hon.), Yale, M.D. (Hon.), Utrecht, Tinsley R. Harrison, M.D., and George B. Eusterman, M.D.; 1949. Chicago: The Year Book Publishers, Incorporated. 7" x 4½", pp. 838, with 138 illustrations. Price: $4.50.

One of the "Practical Medicine Series" of year books.

"An Atlas of Amputations", by Donald B. Slocum, M.D., M.S.; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 11½" x 8¼", pp. 584, with 564 illustrations. Price: £10 10s.

The author's purpose is to present to the reader those methods of the past which have stood the test of time and to add the results of recent experience.

"The Medical Clinics of North America" (issued every two months); 1949. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Mayo Clinic Number. 9" x 5½", pp. 318, with illustrations. Price: £6 (paper binding) and £7 5s. (cloth binding) per clinic year.

This is a Mayo Clinic number. Five papers form a symposium on gastro-intestinal conditions; there are fifteen papers on other subjects.

"Tuberculosis Nursing", by Jessie G. Eyre, S.N.R., B.T.A. (Hons.); 1949. London: H. K. Lewis and Company, Limited. 8¼" x 5½", pp. 308, with 102 illustrations. Price: 21s.

A guide to those who wish to undertake tuberculosis nursing.

The Medical Journal of Australia

SATURDAY, DECEMBER 10, 1949.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE FAR WEST CHILDREN'S HEALTH SCHEME OF NEW SOUTH WALES.

THE Far West Children's Health Scheme of New South Wales is celebrating its silver jubilee. The story of the venture, told in a special issue of the *Far West Magazine*, is one which should be widely known, for the work of the last twenty-five years must extend.

The services provided by the scheme are as follows: (i) An annual seaside camp is set up for far-west children who have never seen the sea. No less than 3000 children have attended these camps. (ii) Baby health sisters travel in the out-back regions; they cover over 30,000 miles each year visiting homes and seeing over 1000 babies each three or four weeks. (iii) A systematic search is made for cripples, and specialists are taken to country centres to examine the children in conjunction with local doctors. (iv) Searches are made for country children needing medical or dental treatment and special attention is paid to children of pre-school age. (v) The costs of transport, treatment, surgical appliances, artificial limbs and so on are provided for necessitous children recommended by country practitioners for special treatment—the services of doctors and hospitals are voluntary. (vi) A transport system is provided by which trained nurses escort patients to and from the country. (vii) A well-equipped home is maintained at which accommodation and treatment are provided for country children; so far 4452 children have benefited by this provision. The scheme originated in 1924 in the mind of the Reverend Stanley Drummond, a Methodist clergyman stationed at Cobar, New South Wales, who was convalescing at the seaside resort, Manly, after a serious illness. He thought that it would be beneficial to the children in the western parts of the State if they could be given a holiday at the seaside. In the following year between 30 and 40 children were brought for a holiday to Manly. From that beginning the work has grown until the services that have been enumerated are now provided. The medical profession became allied with the undertaking when Dr. George M. Barron, then practising at Manly, became medical superintendent. He is chairman of the Council.

In commending this work to the interest and practical sympathy of medical practitioners and other members of the community, we would draw attention to two important facts. First of all the scheme provides a field for voluntary work of a most valuable kind, work that is the more effective and heartening because, being voluntary, it helps to cement a community relationship that is needed more than ever at the present time. In the second place the work is "turning liabilities into assets". Children in need of holiday and change are given both. Cripples are found and treatment is provided for them. The Department of Public Instruction associates itself with the organization and provides educational facilities for children in the care of the scheme. Many "far west children" who were severely handicapped are now useful members of society.

THE EARLY DIAGNOSIS OF LUNG TUMOURS.

IN every paper, comment or discussion on malignant disease, reference is sooner or later made to the supreme importance of early diagnosis. This reiteration is monotonous and even irritating, but irritation is a better response than none in a vital matter. It is sobering to reflect on the comparatively recent statement by Ralph Adams, of the Lahey Clinic,[1] that the ratio of operable to inoperable carcinoma of the lung for the ten years before he wrote had remained almost constant at 1:3. Adams commented that he sometimes wondered if all who could be reached by the written word already knew the value of the point (early diagnosis), and by their actions achieved the early diagnoses that were made, while a larger segment of the profession and the public constantly failed to be reached or forced into action by the available knowledge which would allow an early diagnosis in a higher percentage of cases. His thought is important, though it would be wrong to let it become the basis of a counsel of despair. Another aspect of the question is the fact that even those who will listen need more than merely to recognize the importance of early diagnosis; they should know the relative value of diagnostic methods and findings, so that they will not lean on false supports. This applies very much to lung tumours. For example, the community must be taught to seek early medical advice about persistent symptoms—cough, hæmoptysis, sputum production, chest pain—and many relatively early diagnoses of lung tumours may be made as a result; but a tumour may be well established before symptoms appear. This is added to the fact, stressed by R. H. Overholt and I. C. Schmidt,[2] that despite a considerable reduction over the years, the delay between appearance of first symptom and diagnosis is still too long. Overholt and Schmidt, as well as Adams, advocate annual X-ray examination of the chests of older people, as an extension of the tuberculosis detection campaigns, in which attention has been paid particularly to the younger person. It is believed that in many cases much of the period of silent growth can be saved by this means, even though only a suspicion is aroused which needs to be verified by further X-ray examination, cytological examination of sputum or material aspirated from the bronchi, bronchoscopy or even thoracotomy. Stress has

[1] *The Surgical Clinics of North America*, June, 1947.
[2] *The New England Journal of Medicine*, March 31, 1949.

been laid on radiological diagnosis also by Adrian Lambert,[1] who is particularly anxious to point out that bronchoscopic examination and symptomatology do not form a good basis for early diagnosis. The important phase from the point of view of resectability of most lung tumours appears to be before symptoms and abnormal bronchoscopic appearances occur. This means that in many cases the hope of early diagnosis—and of surgical cure—will rest on exploratory thoracotomy following the finding of suspicious radiological appearances—a formidable diagnostic procedure, surely, albeit considerably tempered by recent advances in chest surgery. The same comment applies to mass radiological methods of detecting early lung tumours as has been made in relation to tuberculosis: they are futile, and even harmful, if facilities are lacking for supplementary diagnostic investigation and treatment. The reasonable argument that with tuberculosis detection is of value in the control of contagion, even if adequate treatment is not possible, does not apply to lung carcinoma. Nevertheless, the practical implications of the matter must be faced. As Overhead and Schmidt put it, "the ability to utilize a lung as a unit of excision" has made it possible to think in terms of cure when that organ is the primary site of cancer; the making of a sufficiently early diagnosis and the provision of effective treatment would appear to depend less on further advances in knowledge and skill than on the application of our present possessions.

A very recent comment on mass radiography, based on extensive personal experience, must be added. George A. Mason,[2] in a review of 1000 cases of carcinoma of the lung, states that despite previous hopes, his experience with mass radiography has been disappointing. No appreciable proportion of patients with operable tumours has come in this way, especially if those patients are excluded who went to the centres because they already had chest symptoms. He refers also to the type of centre at which patients with "suspicious" conditions are held for observation instead of being referred at once for investigation—some in his experience so long that the tumour has become inoperable as a result. He feels that better dividends would be paid by a system in which every patient entering a hospital for any purpose should have an X-ray examination of the chest. This is reinforced by his comment in another place that too many patients probably still linger in the medical wards of general hospitals until all chance of resection has been lost. From his remarks it would seem that Mason, despite his disappointing experience of mass radiography, still looks to the X-ray film as the main factor in early diagnosis; perhaps the factors which have led to disappointment are not insuperable.

[1] *The American Journal of the Medical Sciences*, January, 1948.
[2] *The Lancet*, October 1, 1949.

Current Comment.

MATERNAL OBESITY.

IT is a common, perhaps usual, phenomenon that during pregnancy the mother's body-weight increases progressively in an amount greater than the increase in the fœtus and generative organs together; retention of tissue fluids and improved general nutrition may fully account for this increase, which usually disappears during the puerperium. A different phenomenon from this, less frequent but well recognized, is the development of decided obesity, sometimes during pregnancy, more often after childbirth has occurred. This is of all grades, but is fortunately rarely so severe as in two cases quoted by J. H. Sheldon,[1] in which a weight increase of approximately 125% occurred after two pregnancies; one woman weighed eight stone one pound at marriage, fourteen stone at the end of the lying-in period following birth of her first child, and eighteen stone seven pounds after the birth of the second child; the other weighed nine stone at marriage, gained weight during two pregnancies and weighed twenty stone four pounds at the end of the second. The two women are members of a series of 40 developing obesity during pregnancy, of whom Sheldon has presented a careful study. He found that the increase might begin either during pregnancy or immediately after the confinement, and might occur after all pregnancies or after only one particular pregnancy—when the baby was often a boy. In typical cases there was an extremely rapid initial gain in weight, which was followed by a static phase when the weight remained stationary at its new high level for an indefinite period, though, occasionally there was a later fall. Abnormal pregnancy was somewhat more frequent among these women than among those in a control group. Menstruation was unaffected, returning to normal after parturition. Lactation was normal, though in some cases there was an excess of milk. The stillbirth and neo-natal mortality was raised, and the women produced an excess of large babies. However, the large babies were almost entirely born to those women who developed obesity in the course of each pregnancy; by contrast, small babies were confined to those women who became fat after confinement from a particular pregnancy. Investigation of the families of the obese women revealed that their mothers had suffered from the same condition more frequently than normal, though their sisters were not specially affected, and they themselves showed a tendency to have been unusually large babies at birth. In later life some of these women developed abnormalities of carbohydrate metabolism; two were diabetic and under treatment for this condition; glucose tolerance tests were performed in 26 cases and the curve in six cases was found to be abnormal and of diabetic type.

Sheldon considers it clear that these obese women provide the reservoir from which patients with the "large-baby diabetic syndrome" are recruited, though not all are affected in this way. This syndrome has been fully discussed by J. A. L. Gilbert and D. M. Dunlop[2] and we would refer those interested to their papers. Sheldon draws attention to the important features that women who develop diabetes in later life tend to have given birth to an unusually high proportion of large babies many years previously, and that these women are often obese. There are, indeed, four main points of agreement between the two conditions: obesity, large babies, a raised fœtal-loss rate and the late development of *diabetes mellitus*. In seeking out underlying causes, Sheldon points out that with the exception of the obesity, increased function of the anterior lobe of the pituitary could account for all these features and also for increased lactation, which may or may not be an integral part of the syndrome. It is more difficult to explain the obesity along these lines, and Sheldon feels that the evidence suggests that the other features are "not so much essential parts of the syndrome as accidental by-products of a deeper cause which of itself inevitably leads to obesity, but in any individual may or may not produce these additional features". Experimental work involving damage to the hypothalamus suggests that this "deeper cause" could well be a hypothalamic disturbance. No autopsy records are available to help, and the relevance to humans of animal experiments cannot always be relied upon, so that at present discussion of the mechanism of this condition or conditions is only speculative. Sheldon's hypothesis, however, is that the primary disturbance may be in the

[1] *The Lancet*, November 12, 1949.
[2] *British Medical Journal*, January 8 and April 23, 1949.

hypothalamus, which, perhaps because of heredity or instability during pregnancy and lactation, is apt to suffer a disturbance of its normal control of weight; such a disturbance tends in most cases to be permanent and the weight-gain is thus permanent. In some cases the hypothalamic disturbance produces also an increase in function of the anterior lobe of the pituitary, which results in large babies, a high fœtal-loss rate and excessive lactation, each in about one-third of the cases. The later development of *diabetes mellitus* may be due to this pituitary hyperfunction or may be merely the result of the prolonged obesity. Confirmation or otherwise of this hypothesis, which does not appear at all extravagant, will probably have to wait on the collection and analysis of much larger quantities of clinical material and the consideration of post-mortem findings when they become available.

AN EVALUATION OF PREFRONTAL LEUCOTOMY.

FROM time to time in these columns and in original articles published in this journal, reference has been made to prefrontal leucotomy and similar operations. Towards these rather startling procedures it has been necessary to adopt a cautious attitude, firstly because they are mutilating operations, mostly performed blindly and with little certain knowledge of their exact physiological basis and effects, and secondly because the assessment of a psychological state is difficult and often equivocal, especially if based on a short period of observation. However, it is now thirteen years since Monez called attention to the operation of prefrontal leucotomy. It was taken up by others within a short period, and it seems reasonable to expect that general experience of this and allied procedures should now be sufficient, in both scope and duration, to warrant some conclusions. For the general practitioner and the physician who may have the responsibility of referring patients to the psychiatrist or the neurosurgeon and also of carrying out after care, a recent evaluation of the subject by Harry C. Solomon[1] should be helpful. Solomon is professor of psychiatry in the Harvard Medical School and a member of the advisory committee to the neuropsychiatric division of the Veterans Administration. His article was published by the Veterans Administration as a technical bulletin. Solomon points out that it is not easy to arrive at completely satisfying statistics for a number of reasons: the variation of operative technique with the surgeon; lack of knowledge of the exact amount of medullary substance and cortex that is destroyed; variation in the pre-operative condition of the patient; difficulties of experimental control. The best that can be offered, he suggests, is a general consensus regarding the two major considerations—the benefits from the operation and the defects which follow it. Spectacular improvement, making possible a return to a position of usefulness in society and sometimes ability to be self-sustaining, occurs in perhaps 10% of cases. In about another 15% to 20% of cases the patients are able to live a relatively happy life and be in part self-sustaining. Probably 80% of patients become more contented and have a more satisfactory existence than before operation. The mortality rate varies; it is generally about 3% to 4%, but under the best conditions only 1% to 2%. The impairment of psychological function following operation is difficult to determine. Tests of general intelligence have revealed little. Tests of concrete and abstract behaviour indicate impairment but of slight degree. Personality tests reveal a greater tendency to repeat responses (perseveration), the responses being stereotyped and conventional; in the use of colour, which is a measure of emotional reactivity, there is greater freedom of response and less rigidity. Undesirable reactions may be an unduly carefree attitude (with euphoria), apathy, procrastination, facetiousness, profanity, outbursts of temper and distractability, but these may lessen in a few months. Some degree of impairment is to be anticipated in important characteristics of personality integration, such as judgement, planning for the future, discrimination, laudable ambition, fine sentiments and creative imagination. In 4% to 10% of cases convulsive seizures occur, but usually they are few. Thus some degree of defect follows leucotomy and this must be balanced, in each individual case, against the beneficial results which are equally likely. Collective experience so far indicates that tension, apprehension, fear, concern, worry and agitation are greatly reduced or abolished; that excitement, aggressive and "assaultive" behaviour, emotional outbursts and rage-like exhibitions may temporarily increase and then fade away; that paranoid ideas tend to vanish or to be much attenuated; that obsessive thinking becomes less distracting, and compulsive actions and ritualistic behaviour cease; that long-standing hallucinations may lessen, though they seldom disappear entirely; that anergic states may become states of activity but are more often unchanged, and activity may even be reduced.

Solomon discusses *seriatim* the several symptom complexes or disease categories in which leucotomy is to be considered. It should suffice here to indicate his main points. Suitable candidates for leucotomy, in his view, are those with manic-depressive psychosis and involutional melancholia who are in a chronic state and have not responded to other forms of treatment; those with obsessive-compulsive neurosis of several years' duration who are incapacitated or progressing towards incapacity, provided that they have had the best available psychotherapy and the possibility of remissions is remembered; certain patients with schizophrenic psychosis who have failed to respond to other forms of therapy; those with chronic paranoid psychosis who have not benefited from other therapy; those with psychosis related to chronic encephalitis, but not those with any other organic disease of the brain; those with severe intractable pain due, for example, to tumours, tabetic crises or trigeminal neuralgia, when other procedures have failed; psychotic patients physically unfit for shock therapy or for physical activity and some with suicidal tendencies. The presence of epilepsy is apparently not a contraindication, but a psychopathic personality may be made worse. It should be emphasized that Solomon stresses always the importance of other forms of therapy and the need to assess carefully every individual patient; in no case does he suggest that leucotomy should be undertaken without a careful weighing of all factors involved, and this fact must be borne in mind when the list of possible indications that we have given is read.

A brief reference to Solomon's discussion of the post-operative state may be useful and of interest. He states that after operation, most patients fall into an apparent deep sleep; two or three days later they begin to show interest and may be able to get up. Urinary incontinence is usual and may last for weeks or longer; it is much relieved by attention and insistence on control. Mostly paranoid delusions are much less, and many previously uncommunicative patients become quite affectionate and give relatives an undue impression of recovery. Many are very outspoken, inconsiderate and uncontrolled in animosities and the like; this is usually a passing phase and is probably a good prognostic sign. Physical sluggishness is common but often only temporary. A tendency to overeat is common. Increased sociability and articulateness may improve the doctor-patient relationship. Careful physical and psychiatric after-care is extremely important. Most observers believe that a final satisfactory outcome depends to a great extent on the skill used to rehabilitate the patient. Whether the home or the hospital is the best environment for rehabilitation is still undecided. Like every other aspect of this question it seems that it must be determined in each individual case. Finally, and this should not be overlooked, when the patient returns home the family needs careful advice, continuing guidance and encouragement—a point which might be extended with advantage to many other forms of therapy than prefrontal leucotomy.

The opportunity for psychotherapy and the facilities for supervised activity and gradual, but not forced, reassumption of responsibility make stay in hospital preferable in some cases.

[1] *The Journal of the American Medical Association*, July 30, 1949.

Abstracts from Medical Literature.

PHYSIOLOGY.

Respiratory Response in Acclimatization to High Altitude.

H. RAHN AND A. B. OTIS (*The American Journal of Physiology*, June, 1949) present data which allow the prediction of the alveolar oxygen and carbon dioxide composition as well as the relative alveolar ventilation when man is exposed acutely to any altitude, during the process of respiratory acclimatization at any altitude and when acclimatization is complete. In addition, the alveolar pathways can be predicted and described once man is acclimatized to any particular altitude and is then suddenly exposed to higher or lower oxygen pressures. These predictions were in part verified by an acclimatization study carried on at an altitude of 9500 feet for a three-week period. Evidence is presented for an oxygen pressure threshold of the chemoreceptor drive of ventilation at approximately 100 millimetres of mercury. This is observed in the ventilation curve of people acclimatized to altitude. However, in acute exposures in which the alveolar oxygen pressure is reduced to 50 to 60 millimetres of mercury this hyperventilation response is completely inhibited for at least one hour. This inhibition is explained as a result of the pH rise due to the decreased oxyhæmoglobin saturation. If the alveolar oxygen pressure in acute exposure falls below 50 to 60 millimetres of mercury, immediate hyperventilation occurs. Exposures of more than one hour at 9500 feet result in a lowering of the carbon dioxide pressure exponentially with time. Final levels are reached after three or four days. After respiratory acclimatization to 9500 feet, the respiratory system becomes more sensitive to carbon dioxide. This response was tested by breathing various carbon dioxide mixtures and analysing the alveolar air after breath-holding. Data are presented which show the reduction of breath-holding time during acclimatization to various altitudes. The various findings emphasize that a large part of the respiratory acclimatization to high altitudes is an adaptation to a lowered carbon dioxide tension.

Effect of Mechanical Vibration on the Patellar Reflex.

D. E. GOLDMAN (*The American Journal of Physiology*, October, 1948) reports that experiments on the effects of mechanical vibration on the patellar reflex of the cat confirm previous observations that such vibration produces an inhibition. It is shown that the inhibitory phenomenon is readily explained on the basis of vibratory excitation of the reflex itself. The autonomic nervous system plays no direct part in the phenomenon. It is suggested that the inhibitory phenomenon may underlie certain effects of the exposure of man to mechanical vibration or intense sound. The experiments described are consistent with the following interpretation. Vibration produces a periodic, synchronous stretch reflex, in which many of the muscle receptors are involved. There is thus a slight continuous contraction of the muscle, and tapping the tendon is now ineffective since the reflex arc is in steady use. However, this does not involve all the motor neurons, since muscular contraction is obtainable via other routes, in this case the crossed extensor pathways. Also, it is generally accepted that a role of the sympathetic nervous system is to enhance rather than to inhibit muscular activity. So far as the specific reflex studies here are concerned, there is at this time little evidence of any direct intervention of the autonomic nervous system, although it is known that stretch and other muscular reflexes may be affected by autonomic stimuli. The author is inclined to interpret Loeckle's findings on the cat as artifacts referable to the practical difficulty of handling the vibrator and to the mechanical transmission of the vibratory motion from the artery to the muscle. Also, Loeckle used a fixed anæsthetic ("Pernoston"), which may have had a depressant effect on the reflex mechanism; his observations on the human sympathectomized subject, however, still require explanation. There is no evidence available at present for elucidating this. The author considers that the phenomena described in this paper are of some practical interest in connexion with the exposure of human beings to vibrating machinery *et cetera*, or to very intense sound fields. A number of unconfirmed incidents have at various times been reported in which such exposures apparently produced muscular weakness, especially in the lower limbs, and even led to collapse, without, however, any injury occurring. The observations made suggest that there may well be a genuine physiological basis for these experiences. Further work will have to be done before any quantitative relationship can be established between the phenomena and the mechanical forces producing them.

Myenteric Reflex.

E. BOZLER (*The American Journal of Physiology*, May, 1949) describes the action potentials and movements produced when the intestines of the anæsthetized dog were electrically stimulated. Local stimulation of the intestine produces responses which spread considerably beyond the stimulated area. Under certain conditions the contraction is restricted to the oral side. Such polar responses, which are designated as the myenteric reflex, may consist merely of an increase in the strength of the rhythmic contractions or they may be more prolonged contractions, chiefly of the circular muscles. The most effective stimulus for the myenteric reflex is longitudinal stretching in the small intestine of the rabbit and stroking the mucosa in the small intestine of the dog. Abnormal stimuli such as electric stimuli or pinching often produce ascending and descending contractions. However, electric stimulation always produces a strictly polar response in the proximal part of the rabbit colon, and in other parts of the intestine if the stimuli are close to threshold. Nicotine in low concentrations applied locally prevents the spread of the response from the stimulated area. Local application of acetylcholine in minimal concentrations produces a polar response like that caused by appropriate mechanical or electrical stimulation. It is concluded that a synaptic mechanism consisting of short neurons and conducting chiefly in an oral direction is involved in the myenteric reflex. The results are in agreement with the conclusion that the spontaneous contractions of the intestine are myogenic, but are ordinarily conducted only for a short distance. The myenteric reflex increases this activity on the oral side and thereby propels the contents of the intestine. The reflex response by itself is not propagated. The peristaltic waves depend on the continuous stimulation by the contents of the intestine.

Anæsthesia and Gastric Secretion.

M. SCHACTER (*The American Journal of Physiology*, February, 1949) states that chloralose and chloralose-urethane anæsthesia cause a pronounced secretion of very acid gastric juice in dogs previously equipped with gastric fistulæ and not subjected to any trauma at the time of anæsthesia. Urethane, "Pentothal" and to a much lesser degree "Nembutal" anæsthesia also bring about the secretion of varying amounts of gastric juice of high acidity under similar conditions. The chloralose-urethane phenomenon was especially investigated. This secretion was regularly found to approach a pH of 0·9 and to possess minimal amounts of pepsin and visible mucus, thus resembling the secretion evoked by histamine. It was completely abolished by the traumatic procedure of preparing the gastric fistula at the time of the experiment, and was at times completely prevented by slight mechanical manipulation of the vagi in the neck. In those instances in which the phenomenon was not abolished by exposure of the cervical vagi, it was immediately arrested after section of the vagus nerve. The secretion returned to full intensity within a few days after vagal section, only to disappear again almost completely as time for nerve degeneration elapsed. The secretion was not associated with a reduced blood sugar level, but was regularly abolished by atropine. It was much reduced or unobtainable in chronically vagotomized dogs. The possible role of histamine in the nervous phase of gastric secretion and the possibility of a nervous mechanism inhibiting the secretory cell directly are discussed. Evidence is presented to suggest that the gastric glands may secrete a neutral or weakly acid, non-mucoid juice under certain conditions.

Acute Hypothermia in Guinea-Pigs.

R. E. GOSSELIN (*The American Journal of Physiology*, April, 1949) reports observations made on thirty unanæsthetized mature guinea-pigs during and after severe reductions of body temperature produced by immersing the jacketed animals in ice water. Measurements included those of colonic temperature, ventilation rate (minute respiratory volume), oxygen consumption, and carbon dioxide production, and electrocardiograms were prepared. Regardless of the rate of cooling or rewarming, heart rates varied linearly with colonic temperatures between 23° and 35° C., falling 17·5 beats per minute for each descrement of 1° C. Below 23° C. the drop was erratic and often precipitous. Sinus and nodal arrhythmias, premature beats of auricular and ventricular origins, auriculo-ventricular blocks, and reversible ventricular fibrillation were all noted. The early phases of cooling

were characterized by increased struggling, hyperpnœa, enhanced metabolism and cardiac slowing. When cooling took place at 0·2° to 0·3° C. per minute, maximal ventilation occurred at 35° C. (33° to 37° C.) and averaged 170% of the pre-cooling rate. At this point maximal rates of oxygen consumption also amounted to 170% of the pre-cooling level or 250% of the estimated basal figure. Below 33° C., ventilation and oxygen consumption diminished progressively with temperature, dropping below pre-cooling rates between 26° and 29° C. and approaching zero slightly below 20° C. Relative to the oxygen uptake, however, the ventilation rate (expressed as the oxygen ventilatory equivalent) remained high to the stage of terminal apnœa. Therefore, the progressive fall in oxygen consumption cannot be ascribed to failure of external respiration. Some animals survived briefly at 17·5° C., and others succumbed at as high as 21° C. This large variation in lethal temperature is unexplained. The mechanisms of a hypothermic death remain obscure, but these data suggest that in severe chilling critical circulatory inadequacy often precedes respiratory failure. Guinea-pigs which did not die promptly in deep hypothermia were allowed to rewarm in room air. During rewarming ventilatory and metabolic rates varied widely among the tests, and the temperature of some animals rose much more slowly than that of others. It is postulated that damage sustained during severe chilling impairs the capacity to repay a thermal debt. It was not possible to predict final rates of recovery from vital signs during cooling or even during the early phases of rewarming. Beyond four to six hours after the immersion, relatively little progress was made in raising the body temperature, some animals being left stranded at distinctly hypothermic levels (about 30° C.). The latter died within ten to fifteen hours of the immersion. Slight departures of the heart rate from predictions based on the colonic temperature appeared to signal such impending deaths. Whatever the derangements responsible for these delayed fatalities, it seems probable that they represent controls different from those operating at the lethal temperature during cooling.

BIOCHEMISTRY.

Acetone Metabolism.

E. BOREK AND D. RITTENBERG (*The Journal of Biological Chemistry*, June, 1949) have shown that deuteriocholesterol is formed on incubation of surviving rat liver slices with deuterioacetone, thus establishing that this tissue can metabolize acetone. At least part of the acetone is oxidized to acetate.

Thyreoid in Scurvy.

S. BANERJEE AND N. C. GHOSH (*The Journal of Biological Chemistry*, August, 1949) demonstrated that the types of glucose tolerance curves for each of three groups of guinea-pigs which received a scorbutic diet, a scorbutic diet with methylthiouracil and a scorbutic diet with thyreoid were more or less similar. This indicates that the deficient utilization of glucose observed in animals receiving the scorbutic diet only is not affected by either hypothyreoidism or hyperthyreoidism. The blood sugar level, three hours after the feeding of glucose to guinea-pigs receiving thyreoid gland and vitamin *C*, was significantly higher than that of the control animals receiving no thyreoid gland. This indicates that hyperthyreoidism lowers the carbohydrate tolerance in guinea-pigs. Methylthiouracil, however, had no effect on the glucose tolerance test, a finding which shows that carbohydrate tolerance is not altered in hypothyreoid guinea-pigs. The altered carbohydrate metabolism observed in scorbutic guinea-pigs is, therefore, not due to either hypothyreoidism or hyperthyreoidism.

Phosphorus Metabolism of Brain.

M. REISS *et alii* (*The Biochemical Journal*, Volume XLIV, Number 3) have confirmed previous observations by other workers on the uptake of radioactive phosphorus by the brain after its injection into normal rats; comparative measurements on other organs were made. The effect of hypophysectomy was to bring about a general increase in the absorption of phosphorus, particularly by the brain and the pineal body. Administration of corticotrophic hormone to the hypophysectomized animals, before injection of phosphorus, restored the absorption to a normal level. It was suggested that the increased rate of P^{32} uptake of the brain in hypophysectomized animals was connected with increased carbohydrate utilization.

Metabolic Functions of Biotin.

P. R. MACLEOD AND H. A. LARDY (*The Journal of Biological Chemistry*, June, 1949) have demonstrated that intraperitoneal injection of sodium bicarbonate containing C^{14} into normal and biotin-deficient rats results in a larger C^{14} fixation into the adenine, guanine, arginine, aspartic acid, citric acid and bone carbonate of the control animal than into the corresponding compounds of the biotin-deficient animal. The implications of these results are discussed.

Dietary Influence of Riboflavin.

C. J. KENSLER (*The Journal of Biological Chemistry*, July, 1949) found that limitation of the riboflavin or protein intake but not the thiamine or choline intake reduced both the riboflavin concentration in rat liver and the ability of rat liver slices to destroy the liver carcinogen N,N-dimethyl-*p*-aminoazobenzene. The addition of biotin or adenine to the diet was without effect on either the riboflavin level or the destruction of the carcinogen by liver slices. Data showing the relationship between the riboflavin level in the liver and the ability to destroy the carcinogen are presented. The inclusion of the carcinogen in the diet further reduced the hepatic riboflavin level and the ability of liver slices to destroy it.

Oxidation of Fatty Acids.

E. P. KENNEDY AND A. L. LEHNINGER (*The Journal of Biological Chemistry*, June, 1949) have shown that morphologically homogeneous mitochondria ("large granules"), separated from rat liver dispersions by the hypertonic sucrose method of Hogeboom, Schneider and Pallade, contain essentially all the measurable activity of the liver in the oxidation of fatty acids. Likewise, the integrated reactions of the Krebs tricarboxylic acid cycle are found in this fraction: Esterification of inorganic phosphate accompanies these oxidations in purified preparations of mitochondria. These bodies have insignificant glycolytic activity. "Mitochondria" prepared by other methods involving saline or water-dispersing media are inactive in these reactions, possibly because of osmotic damage.

Glucose Uptake and Glycogen Synthesis.

C. H. LI *et alii* (*Archives of Biochemistry*, July, 1949) have investigated the action of insulin on glucose utilization and glycogen synthesis in the isolated diaphragm from normal and hypophysectomized rats and in rats previously treated with adrenocorticotropic hormone or adrenal cortical extract. The diaphragm of hypophysectomized rats utilizes glucose and synthesizes glycogen to the same extent as does that of normal animals. There is no change in the glucose uptake of the diaphragm of rats injected with adrenocorticotropic hormone or with adrenal cortical extract, but glycogen breakdown occurs in these diaphragms. The insulin effect in promoting glycogen storage is impaired in the diaphragms of animals treated with either adrenocorticotropic hormone or adrenal cortical extract.

Radiobiotin and Carbon Dioxide Fixation.

D. B. MELVILLE *et alii* (*The Journal of Biological Chemistry*, August, 1949) have synthesized biotin, labelled with C^{14} in the ureido carbon atom, from radioactive phosgene and the diamino acid derived from biotin. The stability of the ureido ring system of biotin during the biotin controlled fixation of carbon dioxide by *Lactobacillus arabinosus* was studied. Growth of this organism in the presence of the labelled biotin, under conditions requiring the participation of biotin in carbon dioxide fixation, did not result in any replacement of the C^{14} of the radiobiotin with non-radioactive carbon. It is concluded that the mechanism whereby biotin promotes carbon dioxide fixation does not involve a transfer of the ureido carboxyl group of the biotin molecule.

Biosynthesis of Urea.

S. RATNER AND A. PAPPAS (*The Journal of Biological Chemistry*, July, 1949) have isolated from mammalian liver and partially purified an enzyme system catalyzing the conversion of citrulline to arginine. Evidence is presented to show that in the reaction *l*-citrulline and *l*-aspartic acid undergo an exchange to form *l*-arginine and *l*-malic acid through an intermediary condensation which utilizes adenosine triphosphate and magnesium, and is presumably endergonic. Two separate enzymes are involved in the over-all reaction, one catalyzing the formation of an intermediary condensation product and the second its hydrolysis. Some general enzymatic properties of the system are described. The mechanism is believed to represent the main physiological pathway of arginine synthesis and urea formation.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 29, 1949, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. J. KEMPSON MADDOX, the President, in the chair.

Neurosyphilis.

DR. E. L. SUSMAN read a paper entitled "The Treatment of Neurosyphilis" (see page 829).

DR. E. T. HILLIARD read a paper entitled "The Treatment of Neurosyphilis" (see page 831).

DR. EDITH ANDERSON said that she disagreed with Dr. Susman's statement about tryparsamide. At the Rachel Forster Hospital for Women and Children they had been obliged to rely on it a great deal, because of the lack of facilities for fever-inducing therapy, and had found it invaluable. At the present time it was used as a follow-up measure when the patients could be persuaded to go into hospital. Dr. Anderson said that she had seen relief of the patient's symptoms and improvement in the response to the colloidal gold test follow tryparsamide therapy, provided it was long continued—the length of treatment being an important point. In ten years only one case of optic atrophy had occurred. The visual fields were always examined as a routine measure at the first and seventh injections. The late Dr. S. Evan Jones had emphasized the point to her that a patient who had received treatment for late syphilis and apparently been cured should be kept under observation yearly after cessation of active treatment for at least five years, when a further spinal fluid test should be made. Dr. Anderson thought that the importance of that second test was not realized as a measure of arrest or otherwise of the disease in an apparently quiescent case.

DR. D. N. EVERINGHAM referred to Dr. Hilliard's remarks about electro-shock treatment and the relatively good prognosis in the "demented" type of general paralysis of the insane. Dr. Everingham asked Dr. Hilliard to give some indication of when electro-shock treatment should be used in cases of dementia.

DR. A. T. EDWARDS said that he thought Dr. Everingham had misinterpreted some of Dr. Hilliard's remarks; Dr. Hilliard had said that in cases of dementia the prognosis was worst of all. Dr. Edwards himself had thought that the better the prepsychotic personality, the worse the prognosis. When the prepsychotic personality was good, the disease did not produce results until a fairly large amount of cell destruction had taken place. When the patient had such a personality that he reacted easily to stress and strain, symptoms occurred early in the disease. Dr. Edwards said that before he had left the Mental Hospitals Department of New South Wales, he had been collecting information which he thought would prove that theory. With reference to malarial treatment, he had two points to make. The late Dr. C. Henry, who had had more experience than anyone in the injection treatment of patients, held that many more positive results would be obtained if the three-fold method of injection was used—intradermal, subcutaneous and intramuscular. As Dr. Hilliard had said, Dr. Edwards did indeed remember the days of injecting material to produce some form of pyrexial reaction; even intrathecal injections of horse serum had been given. The procedure had been most frightening to the unfortunate medical officer carrying it out. In pre-penicillin days, at the Mental Hospital, Morisset, they had used the intravenous injection of "T.A.B.", with lumbar puncture and the use of tryparsamide (with apologies to Dr. Susman); at Callan Park they had used malaria with tryparsamide; and at Broughton Hall they had used the "Inductotherm" with tryparsamide. The results of all three methods were practically the same with similar types of patients. The introduction of penicillin had altered the whole course of the disease. Rapid results were obtained with the simultaneous use of penicillin and malaria. Dr. Edwards could not see why one should wait for the result of the malaria treatment to be apparent before administering penicillin; penicillin was without effect on the malaria parasite. Dr. Edwards said that he had not been brave enough to give up tryparsamide. He had had no definite evidence that it had no beneficial effect, and in an otherwise hopeless disease the patient had to be given the benefit of any doubt.

DR. EDGAR THOMSON stressed the importance of the false positive reaction in the Wassermann test. He said that it should be remembered that there was such a thing, and that blind implicit faith in the result of the Wassermann test was a dangerous faith. There were many reasons for false positive reactions; the technical false positive reaction could be avoided by careful technique; the biological false positive reaction could be avoided by careful clinical examination of the patient. It was possibly fair to say that there was only one disease which, like syphilis, produced a persistently positive Wassermann reaction, and that was yaws. Diseases that had been blamed, for example, malaria, leprosy *et cetera*, did not produce a persistently false positive reaction so often as had been stated. However, there was the rare person who gave a positive reaction to the Wassermann test in the absence of syphilis or any of the other diseases mentioned—this was the anomalous false positive reaction and was difficult to assess. A true false positive reaction was variable—present at one time, absent at another. If the reaction persisted and the other diseases had been excluded, then it was fair to assume that the case was one of syphilis. Dr. Thomson said that he could not emphasize too much the fact that false positive reactions did occur, and every effort had to be made to exclude all other possibilities before the person who presented no other evidence of specific disease than a false positive reaction to the Wassermann test was labelled syphilitic. Referring to penicillin, Dr. Thomson said that both speakers had mentioned procaine penicillin, but neither had mentioned the best type of penicillin to use, and neither had said whether procaine penicillin with its slow rate of absorption was better to use than ordinary penicillin. Dr. Thomson asked for information on that point, because he himself was not convinced that procaine penicillin had any advantages over ordinary penicillin except that it saved the patient from repeated injections.

DR. N. E. KIRKWOOD referred to the real difficulty experience even at the present time in obtaining and using malarial blood. He said that the question had been asked whether fever induced by other means was as efficient therapeutically as malarial hyperpyrexia. Often one encountered the difficulty of the patient to whom malaria could not be given. A strain of malaria was obtained, one or two patients would take it, another would not; or the patients would take it and have one or two rigors and then stop. If an "Inductotherm" was available, and one knew that it was as efficient as malaria, it would be a help. Dr. Kirkwood said that he would be glad to know whether artificially induced fever was as good as malarial fever.

DR. W. L. CALOV said that Dr. Susman had said that the Herxheimer reaction might be disregarded, and no other speaker had mentioned it. Dr. Calov had always believed that Herxheimer reactions could occur, and he wanted to know more about the subject. He had rather thought that with the introduction of penicillin, which was a much more potent anti-spirochætal agent than any of the other materials used, Herxheimer reactions would be much more severe and much more frequent. Dr. Calov asked for information.

DR. S. G. SANDES had a word to say on the vexed question of tryparsamide. He said that he had dealt with a considerable number of cases of neurosyphilis during ten years or so, and looking back he could think of a number of patients with visual disturbances; if the tryparsamide treatment was discontinued early, some recovered fully, others not so well. Two patients became completely blind on ordinary dosage after the third or fourth injection. Treatment with penicillin and malaria or more modern methods of hyperthermia should replace tryparsamide. Dr. Sandes agreed with Dr. Susman that the proper place for tryparsamide at the present time was in the waste-paper basket.

Dr. Maddox, from the chair, said that he agreed with Dr. Thomson that absolute reliance on the positive Wassermann reaction had led one astray from time to time. He thought that it was only during the years between the two World Wars and the years of the second World War that faith in the infallibility of the serological reaction had been shaken to some extent. In the older days there must have been a number of people considered to be suffering from neurosyphilis and other forms of syphilis who were vigorously treated with metallic compounds unjustifiably. In the field of syphilis one had to pay homage to the vast American experience. The Americans had had a tremendous field in which to try all kinds of permutations and combinations of therapy. Dr. Maddox understood that in general their present conclusions were in favour of the use of penicillin alone. One authority had told him in conversation that the main thing in the treatment of neurosyphilis was "never to rest on one's oars". Other antibiotics might ultimately be found to be superior to penicillin, but at the University of California they were loath to abandon their experience in hyperpyrexia induced by a simple form of "hot box"; they

did not use the stream-lined cabinet, but an apparatus of blanket and hot lamp type that would be easily reproducible in country centres in Australia. With regard to the intrinsic value of malaria as opposed to artificial forms of hyperpyrexia, Dr. Maddox said that he had been interested to hear what the speakers had to say. It had never been proved that malaria had any intrinsic virtue over other forms of hyperpyrexia. There appeared to be a controversy over the necessity for arsenic, and over the form of arsenic to be employed if the need for it was accepted. Naturally physicians whose experience went back twenty years had been loath to give up such an old friend as arsenic, just as Dr. Robert Scot Skirving had no doubt been loath to give up using his silver pills.

Dr. Susman, in reply, said that his withers were unwrung by the fact that tryparsamide was still being used at the Rachel Forster Hospital. It was a dangerous drug, and, in his view, its use was never justified. Dr. Susman regretted that no mention had been made in the discussion of spinal syphilis. In his experience syphilitic meningo-myelitis was a fatal disease. He had treated six patients so affected since the end of the war, and all had died. They had all become spinal animals, and died of bed-sore sepsis or ascending urinary infection. Some had been treated conservatively and timorously, others vigorously. In such circumstances the Herxheimer phenomenon was a real problem, and one did not know what to do for the best. There was nothing specific about malarial therapy. It was the fever that was the therapeutic agent. It did not matter much how the fever was produced. Malaria was usually the most convenient method.

Dr. Hilliard, in reply to Dr. Everingham, said that electro-convulsive therapy could be expected to have its greatest success in the depressed types of *dementia paralytica*. In reply to Dr. Edwards, he agreed that the intradermal injection of malarial blood was as successful as other methods of injection. For rapid results a combination of penicillin and malarial therapy was most favourably commented on. In reply to Dr. Thompson, he suggested that the use of procaine penicillin was more comfortable to the patient, but that it was too early yet to compare its end results with that of penicillin. In reply to Dr. Kirkwood's question whether the end results of inductotherm were as good as that of malaria, he said that the figures of the Health Department showed that that was so. Dr. Hilliard pointed out that one speaker had spoken of unfavourable results with tryparsamide, whilst another speaker had stated that favourable results and no undesirable symptoms had occurred over a period of years.

Dr. Maddox, from the chair, said that he was glad that Dr. Hilliard had mentioned the name of Dr. Prior, who had done a great deal in New South Wales towards establishing the local experience in hyperpyrexia. The discussion had well crystallized the present situation. Five years had scarcely passed since the introduction of the present range of penicillin therapy. The current views had been well summarized, and those present were grateful for the contributions of Dr. Susman and Dr. Hilliard.

A MEETING of the New South Wales Branch of the British Medical Association was held at Saint Vincent's Hospital, Sydney, on October 20, 1949. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staffs of the hospital.

Complications following Head Injury.

DR. DOUGLAS MILLER showed a series of patients with complications following head injury. The first was a young man, who after a head injury suffered from chronic headache for some weeks, and progressive mental deterioration. On examination of the patient, there was some papilloedema, but no other abnormal signs were found. A plain X-ray film of the skull showed a huge aerocele in the left frontal region; at operation this proved to be within the substance of the left frontal lobe and communicating through a channel of adherent brain and opened dura with an anterior ethmoidal cell. It had been repaired by *fascia lata* graft, the patient had made a very good recovery, and he had resumed his work as a university student.

Another patient shown had had a head injury in 1945, which was of a severe degree, having left him totally deaf. After that head injury, he had had four very severe attacks of cerebro-spinal meningitis at intervals of several months, in each one becoming deeply comatose. Repeated X-ray examinations had revealed no fracture in the orbito-ethmoidal region, but eventually it had been possible to detect a small fracture running out into the right frontal sinus. Exploration showed an adherent tear in the dura at this region and a small open communication between the sinus and the subdural space. That had been repaired by fascia graft, and he had made a good recovery and had had no further attacks of infection.

A third patient had suffered a head injury and, while in hospital, had a severe attack of meningococcal meningitis. It had been possible by special X-ray examinations of the ethmoidal region to demonstrate a fracture in the roof of the anterior ethmoidal cells, and at operation that had been found to communicate through an open dural wound with the subdural space. It had been patched with a *fascia lata* graft.

Another patient with chronic subdural hæmatoma was shown. The patient had become drowsy some days after a head injury and no relevant physical signs were found. His head was explored on both sides and an extensive subdural hæmatoma evacuated from the left side. The cavity had been filled with air, and X-ray photographs showed the extent of the subdural hæmatoma. Dr. Miller commented on the fact that though the hæmatomata might be, and usually were, of such great size, they were rarely associated with any lateralizing or localizing signs.

Tic Douloureux.

Dr. Miller showed two patients with *tic douloureux*, who had undergone operation in the form of intracranial division of the sensory root of the trigeminal nerve. The patients both had long histories of intense pain and demonstrated well the characteristics of the complaint: firstly, that it was always anatomically within the trigeminal field; secondly, that it was spasmodic, lancinating and intense; thirdly, that it could usually be started by a light touch over the various trigger zones; and fourthly, that the patients lived an almost incredibly terrible existence. Dr. Miller commented that it was an extraordinary fact that though proper surgical treatment was always readily available to the patients, most of them appeared to go months or years before being subjected to surgical attack. He mentioned that the risks of the operation were negligible, though many medical men and patients seemed to have surrounded them with an aura of a totally unjustifiable fear.

Pineal Tumour.

DR. J. L. DOWLING showed a young boy, who had a short history of intense headache and unsteadiness on his feet. Investigation had shown him to have a pineal tumour. He had an enormous hydrocephalus and very high grade papilloedema. The Torkildsen operation, which consisted of leading a tube from the lateral ventricle into the *cisterna magna*, had been performed and he was having deep X-ray treatment. The Torkildsen operation had had the effect of bringing down his intracranial pressure, and his papilloedema had subsided. He was at the time of the meeting quite symptom-free, except for some limitation of the upward movement of the eyes. It was pointed out that pineal tumours were usually slow-growing and were particularly radiosensitive. The problem had formerly been to overcome the mechanical block caused by the tumour at the posterior end of the third ventricle. Attempts at radical removal of the tumour had invariably been fatal. The ingenious operation devised by Torkildsen had overcome the difficulty; with it the mechanical obstruction could be relieved, and the tumour was usually well controlled by radiotherapy.

Carcinoma of the Stomach.

DR. V. J. KINSELLA presented two patients with carcinoma of the stomach to show that operation was worth while for the condition. The first patient had undergone gastrectomy in November, 1945, and was in good health at the time of the meeting. The second patient had undergone gastrectomy, with removal of half of the ascending colon, the transverse colon and half of the descending colon, for an adherent growth, in February, 1947; he was in good health at the time of the meeting.

Extrapleural Transthoracic Gastrectomy.

Dr. Kinsella then presented a female patient, who had had a partial gastrectomy on July 30, 1948, because of hæmatemesis from a large gastric ulcer, which had eroded the splenic artery. During induction of local anæsthesia the hæmorrhage had become catastrophic and the abdomen swelled visibly in a few minutes. The erosion in the splenic artery had become a rent from which the blood issued in a strong and "audible" jet (its "audibility" was affirmed by Dr. R. Condon). Bleeding was controlled and partial gastrectomy performed. On account of fixity of the stomach and concealment of the left gastric artery in an ulcer mass, as well as of the poor condition of the patient (on admission

to hospital she had a hæmoglobin value of 20%, and 300,000 red cells per cubic millimetre), the cardiac portion of the stomach remaining after gastrectomy was not mobilized, but the ulcer edge was used for the anastomosis. The patient recovered and progressed well for some months, but then returned with a high anastomotic ulcer. On August 30, 1949, the remaining cardiac end of the stomach was removed and an œsophago-jejunostomy "in—Y" was performed. The approach was through the left side of the chest, after removal of the tenth and eleventh ribs, the pleura being pushed up but not opened, and a circumferential incision being made in the diaphragm. That gave a very satisfactory exposure. The patient was well at the time of the meeting, apart from some difficulty in swallowing solids due to a narrowing at the œsophago-jejunostomy opening. That was to be dilated by bougies.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Lecture Theatre, Frome Road, Adelaide, on November 4, 1949.

Clinical Interpretation of Vitamin B Excretion Tests.

MISS J. CLELAND, in a paper on the clinical interpretation of vitamin *B* excretion tests, said that methods for estimating urinary thiamine, riboflavin and methylnicotinamide content which did not require expensive apparatus and were not time-consuming were being used at the Institute of Medical and Veterinary Science for routine estimations. The amount of thiamine was estimated by its conversion to thiochrome by potassium ferricyanide and extraction of the thiochrome with isobutanol. A blank tube was prepared in the same way, but with the omission of the ferricyanide, and to that blank tube standard thiochrome was added until the blue fluorescence under ultra-violet light matched that in the unknown extract. By that means allowance was made for any non-specific fluorescence. Salicylates, which gave a similar but more intense fluorescence than thiochrome, interfered with the test and had to be excluded. Methylnicotinamide would also interfere with the estimation if present in high concentration (as after nicotinic acid therapy), but was not normally troublesome. Statistical analysis of the results obtained in the last two years had shown that significantly lower values than normal were not uncommon, and were found most frequently in alcoholics or in people with peripheral neuritis, congestive cardiac failure, cirrhosis and cancer of the liver, or with symptoms or history of dietary deficiencies. Normal values were obtained in cases of arthritis and rheumatism. No association was found between achlorhydria and hypochlorhydria and the excretion of thiamine, but low values for thiamine were frequently found in cases of gastro-intestinal disturbance from any cause. That might merely reflect poor choice of food, which often resulted from the dyspepsia, and be accentuated by interference with absorption. Patients with diseases of the liver were divided into two groups, those with a normal serum phosphatase content (mainly those with cirrhosis or cancer) and those with a raised serum phosphatase content (chiefly obstructive jaundice and infectious hepatitis). The latter group gave normal values; the former had reduced excretion of thiamine. Values from those with congestive cardiac failure fell into two groups, one showing particularly low values and the other values within the normal range. It was significant that all except two in the latter group were receiving treatment with mersalyl. The greater excretion in those cases did not appear to be solely due to diuresis, and it was hoped that further work could be done in the group. By far the largest number of cases came from people with peripheral neuritis, many of whom had lowered thiamine excretion. It was gratifying to find that in a high proportion of the patients with normal values some other cause of the neuritis (such as arsenic poisoning or disk lesions) was found. In that group, too, lower values were obtained in summer than in winter—possibly a reflection of a change in diet in the hotter weather. Efforts were being made to see if there was any correlation between kidney function tests and vitamin *B* excretion.

The amount of riboflavin was estimated by extracting the riboflavin in butanol : pyridine after preferential oxidation with permanganate. A blank tube was prepared by destroying the riboflavin in an aliquot of the extract by exposure to sunlight, and to that a standard riboflavin solution was added until the fluorescence under ultra-violet light matched that in the unknown. Roughly 40% of the values found in abnormal urine were below the range found for forty normal people, but as the method had been in use only for a few months there was as yet insufficient material for statistical analysis.

Methylnicotinamide formed a highly fluorescent compound by the action of acetone, and a blank tube could be prepared by omitting the acetone. Reduced excretion was found less frequently than in the case of thiamine, but nevertheless significantly lower values were found in alcoholics and in patients with peripheral neuritis, gastro-intestinal diseases or dietary deficiency, but did not differ significantly from the normal in people with rheumatism and arthritis, congestive cardiac failure or diseases of the liver (with or without raised serum phosphatase levels). It was hoped that the estimation of the compound might be used as a means of assessing the methylating capacity of the liver and thus be an additional means of detecting liver dysfunction. However, no difference was found between the ability of normal people and those with cirrhosis or cancer of the liver or infectious hepatitis to methylate test doses of either nicotinic acid or nicotinamide.

Miss Cleland considered that the methods described had proved of value in clinical practice, particularly in those cases of so-called conditioned deficiencies in which diet alone would not explain symptoms which could be due to lack of the vitamin *B* complex.

Amino Acid Metabolism of Bacteria and Antibiotic Action.

MR. P. A. TRUDINGER discussed recent advances in the amino acid metabolism of bacteria and the action of some antibiotics. He traced the correlation between synthetic ability and nutritional requirements of bacteria and pointed out that there seemed to be a rough comparison between increasing dependence on external nutriment with, firstly, increasing parasitism and, secondly, the change from Gram-negativeness to Gram-positiveness. Examples of nutritional requirements of several groups of bacteria were given to illustrate the point. It was stated that concurrently with decreasing synthetic ability there was also a decrease in catabolic activity. The deaminases and decarboxylases were discussed briefly.

The work of Gale on the amino acid assimilation by bacteria was reviewed. The following particular points were made. Gram-positive but not Gram-negative cells concentrated certain amino acids in their internal environment to various degrees. In *Streptococcus fæcalis* lysine passed through by simple diffusion, but glutamic acid only in the presence of concurrent fermentation; however, both amino acids required energy to pass out of a cell. Synthetic detergents and tyrocidin affected the bacterial cell wall, so that amino acids were liberated immediately. Dyestuffs of the triphenylmethane series inhibited the uptake of glutamic acid into the metabolic pool, but not its assimilation. Cells grown in penicillin-containing media failed to assimilate glutamic acid; that fact might be correlated with the effect of penicillin on nucleic-acid metabolism. Sulphathiazole added to growing cells apparently prevented the uptake of glutamic acid into a peptide linkage. The significance of these results was discussed in connexion with chemotherapeutic activity.

Correspondence.

SOME PROBLEMS ASSOCIATED WITH THE MANAGEMENT OF CARCINOMA OF THE BREAST.

SIR: Mr. V. J. Kinsella's letter in your issue of November 12, 1949, is apparently an attempt by an inquiring mind to assess the true value of radiotherapy in association with surgery in the treatment of operable cases of carcinoma of the breast. I feel that he has contributed little except confusion to the matter, and it is unfortunate that he concludes "the present evidence suggests that routine prophylactic pre- or post-operative radiotherapy in carcinoma of the breast is an unjustifiable gesture, nearly always futile, sometimes harmful and rarely useful".

It is astounding that Mr. Kinsella asks: "Has there been any fundamental improvement in radiotherapy over the last fifteen years?" It appears that Mr. Kinsella is quite out of touch with the advances in this field. During my career as

a radiotherapist, there have been revolutionary changes in radiotherapy, in technique in the estimation of tumour doses, and the knowledge of factors influencing the biological effect of radiation. I am tempted to quote from the last words of another letter below that of Mr. Kinsella's and also discussing a review of his on another subject, that his views are "obsolete, inaccurate and grossly misleading"—and especially to the general practitioners who have the primary care of these cases and have to refer them for treatment. Or is Mr. Kinsella merely being provocative with his tongue in his cheek?

My answer to his question concerning comparative doses delivered to the sterno-clavicular joint is that the estimated dose in this area in my post-operative technique is the same as that in cases given pre-operative deep X-ray therapy, or deep X-ray therapy alone. To the same area affected by a metastasis, it would seem sound practice in most instances to irradiate a wider field than just the immediate vicinity of the metastasis, and in that case my dose would be the same as in the technique mentioned above. Occasionally a small field might be used, and then a somewhat larger dose (not very much greater, as Mr. Kinsella says) might be given, because it is tolerated; but I fail to see how he can deduce from this that my other doses are futile and have no effect whatever. I can assure him that I have successfully removed all clinical signs of carcinoma for over five years in cases in the inoperable stage with extensive regional involvement using the modern technique referred to above.

Mr. Kinsella states that it is impossible to give a desirable dose in treating the breast and associated lymphatic areas. If he means a lethal dose in all cases, I agree with him. It is well known that breast cancer shows a fairly wide variation in radiosensitivity from case to case, the majority being of intermediate sensitivity; but surely because of some resistant cases, one does not deny radiation to others? Mr. Kinsella appears to decide, quite unreasonably, on the basis of one case of recurrence in an irradiated area, that prophylactic radiotherapy is irrational. He also states that radium has been tried and abandoned in breast cancer. It is true that radium has been superseded in many clinics by deep X-ray therapy because of modern improvement in the latter. It is, however, still used in several of the leading centres. I also use it myself in selected cases where its particular technical advantages are of value.

An appreciable percentage of the total cases attending any radiotherapeutic clinic are cases of carcinoma of the breast, and quite frequently those showing recurrence after radical surgery. It is more than a pity that Mr. Kinsella does not attend to see for himself the number of recurrences with which I and my fellow radiotherapists have to deal in our out-patient clinics. Presumably Mr. Kinsella does not advocate any attempt to prevent these by radiotherapy. Several of my friends in the surgical world in Australia have expressed to me in the past concern at the bad results from radical surgery alone, and the hope that a new approach might be found. The figure for the percentage of regional recurrences after radical surgery, in the best hands, has been given as 25% to 40%. Haagensen (a surgeon for whom I have the utmost admiration) has enunciated strict criteria for operability. Cases not conforming to these he sends for radiotherapy alone. It is a pity that such strict criteria are not always followed. Incidentally, it may be of interest to note that Haagensen's operation for this disease is very thorough and takes some five hours to perform.

During my overseas trip last year, I visited Great Britain, Sweden, France and America, and I found almost general advocacy of prophylactic radiotherapy in this disease. The standard of radiotherapy in Great Britain is very high, because, perhaps more than anywhere else, sound principles founded on advances in radiobiology, pathology and physics have been laid down, and followed in all centres. It seems significant that Mr. Kinsella quotes some American results to discountenance those from Great Britain.

McWhirter, whom I know, and with whom I spent some time during my trip, was primarily surgically trained and is now Professor of Radiotherapy at Edinburgh. At a meeting of the surgical staff in 1941, at which he took the chair, it was unanimously agreed that the routine method of treatment in most cases of carcinoma of the breast should be simple mastectomy, *plus* deep X-ray therapy. This has been followed ever since, and still the surgeons there see no reason to change. The records are meticulously kept and refer to practically all cases in south-east Scotland, and include all advanced and even moribund cases. The survival rate at five years of all "operable" cases (941) for the new method of treatment is 55·9% for the period 1941-1945; and for all cases for the same period (1146) 50·1%. Compare this with the figures for five-year survival in operable cases after radical mastectomy alone for 1930-1934, that is, 35·6%, and the similar figure for operable cases for 1935-1940 after radical surgery and post-operative radiotherapy, that is, 44·0%. McWhirter's results have created enormous interest in Europe and America, and when I saw him he had just returned from a tour as a guest lecturer in America. It seems hardly wise to dismiss his work therefore with a shake of the unbelieving head.

With respect to lymphœdema of the arm following radiotherapy, I can only say that I have rarely seen it with modern technique, and then only when it could be fairly attributed to carcinomatous infiltration. McWhirter assured me that he never saw it with his method of radiotherapy of the axilla, and I can verify this from the number of cases I saw in his clinic with normal-sized arms and free movements.

Mr. Kinsella is, no doubt, aware of the work of Handley at the Middlesex Hospital, London, in showing that approximately 20% of stage I cases have involvement of the internal mammary lymph glands. I was also informed by Dr. Neilsen, of Copenhagen, that surgeons in Denmark had shown that a similar percentage with involvement of the supraclavicular glands (as shown by biopsy) occurred in stage I cases.

These facts make one wonder whether so-called stage I cases should not have irradiation of the internal mammary and supraclavicular lymph glands in the absence of any other efficient method of treatment, although they are frequently denied it. There is a vast amount of evidence that the results in stage II cases are appreciably improved by post-operative deep X-ray therapy.

When in New York, I attended, at the Presbyterian Hospital, a lecture, under the chairmanship of Dr. Haagensen, by Dr. Engelstadt, of Oslo, and from a series of 1384 cases of carcinoma of the breast, he quoted a five-year survival rate of 69% with operation and post-operative irradiation.

An urgent necessity in Australia, and one which I have persistently advocated, is the establishment in the main centres of a first-class records and statistical department, to cover the follow-up and statistics of all cancer cases in the region (or at least the principal general hospitals) whatever the treatment. Results quoted from highly selected groups of cases are of little value; and the expression of mere opinions does untold harm.

Yours, etc.,

135 Macquarie Street, HAROLD J. HAM.
Sydney,
November 21, 1949.

THE MENTAL LIFE OF THE CHILD.

SIR: According to a review in your issue of November 12, the author of a book, "The Mental Life of the Child", is a firm believer in "the reality of the infantile wish to return to the mother's womb". I would venture to suggest that the innate dread of snakes which most of us possess is actually a memory of the aggressive and wriggling spermatozoon attacking and piercing the defenceless ovum.

Yours, etc.,

The Hall, W. A. OSBORNE.
Kangaroo Ground,
Victoria.
November 16, 1949.

THE TREATMENT OF STATUS EPILEPTICUS.

SIR: In view of your article in "Current Comment" on the treatment of *status epilepticus*, it may be interesting to note that for the past five years paraldehyde has been the mainstay of this hospital in the treatment of this condition.

Our technique varies from that described in *The Lancet*, in that two to three cubic centimetres (depending on the weight of the patient) are given intravenously. Occasionally a second dose needs to be given, but in most cases the one administration terminates the status. The only feature which is liable to perturb those not accustomed to the treatment is the occurrence of laryngeal spasm at the time of injection. This may occasionally be very marked, but is always brief and in our experience has never led to serious consequences.

Yours, etc.,

ISABEL WILLIAMS, B.A., M.R.A.C.P.
Lachlan Park Hospital,
New Norfolk,
Tasmania.
November 18, 1949.

THE ORIGIN OF MALIGNANT DISEASE.

SIR: While research has given us a great wealth of information concerning malignant disease, the origin of the condition remains, in general, a dark mystery.

I should be interested if one of your correspondents could inform me whether carcinoma has ever been investigated as a possible late manifestation of some comparatively simple condition. If the transient and painless ulcer of primary syphilis can, in time, produce all the woes of *tabes dorsalis* or general paralysis of the insane, could not carcinoma be a late result of some other condition such as subclinical poliomyelitis, Vincent's infection or even a long-forgotten wart?

Yours, etc.,

Yeoval, BARBARA J. BURFITT.
New South Wales,
November 17, 1949.

Obituary.

JOHN AUSTIN CAHILL.

THE death of Dr. John Austin Cahill, which was recorded in these pages, has been a heavy blow to the Victorian Branch of the British Medical Association. His greatest service to the community was given in his general practice at Fitzroy, where he typified what is best in medicine, putting service first and self last. Of what he did for the Victorian Branch as Treasurer Dr. W. F. Simmons and Dr. David Roseby have written in their personal appreciations. But Cahill also served the Branch as President in 1943; before this he had been a member of the Council since 1930. At its meeting on August 24, 1949, the Council adopted the following resolution:

> That the Council of the Victorian Branch of the British Medical Association records with regret the death of John Austin Cahill, M.B., B.S., who was a member of the Council from 1930 until his death and whose appointments as President of the Branch in 1943, Honorary Treasurer in 1946 and as a Trustee of the Medical Society of Victoria in 1947 reflect the esteem in which he was held by his colleagues.

Cahill was a member of the Council of the Medical Defence Association of Victoria and Chairman of Directors of the British Medical Agency of Victoria, Proprietary, Limited. In 1943 he was elected a member of the Australasian Medical Publishing Company, Limited. His president's address to the Victorian Branch, published in this journal in January, 1944, dealt with the housing problem and showed that he recognized bad housing as one of the great causes of ill health. John Cahill was a friendly soul—people not only esteemed him for the work he did for them, but they liked him as a man. He was quiet and unassuming, generous and hospitable; those who knew him and worked with him will miss him in many ways. The sympathy of the medical profession from many parts of the Commonwealth is extended to his wife.

Dr. W. F. Simmons writes: When the news of the death of John Cahill reached me I was stunned, for only that morning I had received a report which cheered me and made me feel it would not be long before he would be able to go home and later have a long holiday before resuming work.

Commencing his medical course when most men of his age would be well established in practice, by diligence, hard work and self-denial and ably assisted by his wife, he graduated in 1918 when well over thirty years of age.

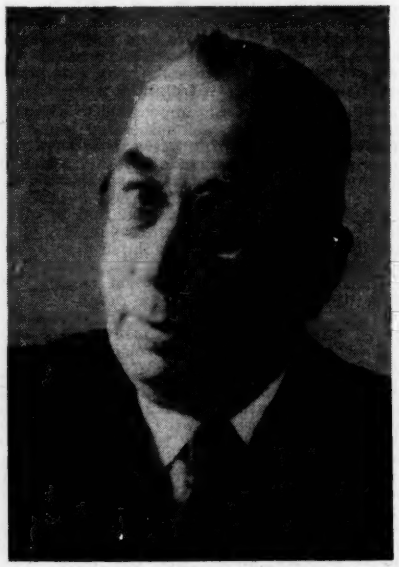
After the usual period of hospital residence he decided to settle in the city of Fitzroy as a general practitioner. Living in an area where there were many tenements and substandard houses, he gave much thought and time to the study of housing, and as medical officer of health of that city undertook in 1937 a world tour to study the latest developments in slum clearance and housing projects.

Realizing that housing and nutrition were the most important contributing factors to positive health, he was particularly interested in the question of pure milk supply and distribution, and as a result of his knowledge of this problem was appointed by the State Government to the Board, as the medical member, which dealt with this matter. For a busy general practitioner, with other interests as well and his honorary work for the British Medical Association taking a great deal of his time, it shows the manner of man he was.

But it is as a visitor to Melbourne that I wish to pay my tribute as a friend to the simple, big-hearted, generous man who kept open house for those who accepted his hospitality. John Cahill was seen at his best in his home where his quietly efficient, warm-hearted wife delighted to honour her husband's friends. On my many visits to Melbourne I have spent many happy hours with them both and I know of many others who have been charmed with their hospitality.

As a fellow treasurer, I have learned to value his advice, for his fifteen years in the business world of Melbourne must have made him an invaluable executive officer of the Victorian Branch and the Medical Society of Victoria. When dealing with the business side of things as they affect the profession, is there any honorary officer of the association in Australia who has, at the one time, held so many appointments?

Professional freedom, the highest standard of medical care and the good name of the association were principles worth



fighting for, and the Federal Council of the British Medical Association in Australia had no more worthy champion of its aims than John Cahill.

To his wife, his brother and his nephews, on behalf of his many interstate friends I wish to offer our deepest sympathy.

Dr. David Roseby writes: It is with a deep sense of personal loss that we of the Victorian Branch record the death of our treasurer and friend, John Cahill, from coronary insufficiency.

It is not given to general practitioners to have recorded of them that they held a place among those who occupy high positions in the scientific and technical branches of our profession. It is in their ranks that the intimate contact between their patients and themselves can be maintained to the full. It is here that the "doctor-patient relationship" makes its presence felt.

John Cahill, known as "Pop Cahill" to students of his year, left a good position at Dalgety's at an age when others would have looked forward to a business career. When he became President of the Victorian Branch in 1933, he recalled a conversation he had had with a prominent member of the firm on resigning from Dalgety's, when he was told that he was too old to become a successful doctor.

Established in Fitzroy, John became the medical father of his flock. He was intensely religious and showed to faiths other than his own a degree of tolerance which was greater than that shown by most men. I was associated with him since his advent to the Council and not once did I hear any word of offence. He was kindness itself and he burnt himself out in his devotion to a large lodge practice.

He came to the Council when we were saddled with a big debt on our building in Albert Street, a debt that

previous finance committees should have met. By his association with Mr. Edgar Ward, our Finance Secretary, he suggested a plan for its liquidation so that it has now almost disappeared.

I saw him twenty-four hours before his death and he was looking forward to a further term as treasurer, though his devoted wife had long warned him of the strain of late meetings of executive, council and finance committees and meetings of the Hospital Benefits Association. We were fortunate to have as treasurer a man of his financial experience, because he could see more easily than any of us the solution of financial troubles.

I have been asked by a large number of his friends, both lay and medical, to express their sense of bereavement at the passing of a Christian gentleman, firm friend and respected member of our profession.

MARIE BROWN.

WE regret to announce the death of Dr. Marie Brown, which occurred on November 21, 1949, at Adelaide.

Public Health.

DIAGNOSTIC CONSULTANTS IN POLIOMYELITIS IN NEW SOUTH WALES.

THE following letter has been received from the chairman of the Hospitals Commission of New South Wales.

Sir: In connexion with the organization to deal with a possible outbreak of epidemic poliomyelitis, the Government has appointed a team of ten physicians in the City of Sydney and three in Newcastle to act as diagnostic consultants in this disease to private practitioners who are free to select any consultant from those appearing in the list attached in any case in which a diagnosis of poliomyelitis may be suspect or in which diagnosis may be difficult.

The cost of this consultant service will be borne by the Government, and the British Medical Association has approved of the conditions of payment.

It is desired that you publish this list in order that it may be known to practitioners that these consultants are available, with a request that they make use of this service in order that they may be fully assisted; and that the hospitals may, as far as possible, be assured that only definite cases of this disease will have to be admitted.

Yours, etc.,
ALAN B. LILLEY, M.B., Ch.M.,
Chairman.

The Hospitals Commission of New South Wales,
Box 3944V,
G.P.O.,
Sydney.
November 25, 1949.

List of Diagnostic Consultants.

The Royal Prince Alfred Hospital:
Dr. C. G. McDonald, 143 Macquarie Street, Sydney. Telephones: BU 2071, FU 7982.
Dr. W. P. MacCallum, 143 Macquarie Street, Sydney. Telephones: BU 1149, FM 4266.

Saint Vincent's Hospital, Darlinghurst:
Dr. Bruce Hall, 185 Macquarie Street, Sydney. Telephones: BW 9044, XB 5013.
Dr. P. J. Markell, 193 Macquarie Street, Sydney. Telephone: BW 7126.

Sydney Hospital:
Dr. A. B. Hogan, 139 Macquarie Street, Sydney. Telephones: BW 4541, WL 1337.
Dr. F. Ritchie, 225 Macquarie Street, Sydney. Telephone: BW 9071.

Royal Alexandra Hospital for Children:
Dr. S. G. Bradfield, 225 Macquarie Street, Sydney. Telephones: BW 9320, UY 1083.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 19, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.[2]	Australia.[2]
Ankylostomiasis	*	4(3)	..	..	3	..	..	..	7
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1(1)	1(1)	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	1	..	..	..	..	..	1
Diphtheria	9(5)	4(4)	3(3)	..	..	..	..	..	16
Dysentery(*b*)	*	3(3)	..	1(1)	..	..	..	..	4
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	2(1)	*	*	..	..	2
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	1	*	*	..	*	1
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*c*)	*	(*c*)	1(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	1(*c*)
Measles	*	*	*	40(17)	*	*	..	..	40
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(6)	11(4)	1	29(21)	..	1(1)	..	..	50
Psittacosis	*	*	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*h*)	*	*	4(3)	*	3	*	..	..	7
Scarlet Fever	39(20)	26(12)	6(4)	6(3)	19(19)	1	..	..	97
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1	*	1(1)	*	*	*	2
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	37(33)	20(14)	14(9)	6(6)	11(4)	1	..	..	89
Typhoid Fever(*e*)	1	..	2(1)	..	..	..	..	..	3
Typhus (Endemic)(*f*)	..	..	1(1)	..	..	..	..	..	1
Undulant Fever	..	..	..	..	1(1)	..	..	..	1
Weil's Disease(*g*)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	41(11)	*	*	..	..	41
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944–1945. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory and Australian Capital Territory.
* Not notifiable.
(*a*) Includes "Mossman" and "Sarina" fevers. (*b*) Includes amœbic and bacillary. (*c*) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (*d*) Includes all forms except in Northern Territory, where only pulmonary tuberculosis is notifiable. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Cases reported include scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease. (*h*) Notifiable disease in Queensland in females aged over fourteen years.

Dr. Gertrude S. Gelkie, 149 Macquarie Street, Sydney. Telephones: BW 8616, JX 2109.

The Royal North Shore Hospital of Sydney:

Dr. I. A. Brodziak, 231 Macquarie Street, Sydney. Telephones: BW 9910, FU 2005.

Dr. Douglas Anderson, 185 Macquarie Street, Sydney. Telephones: BW 6944, XF 1193.

The Royal Newcastle Hospital:

Dr. C. A. Clark, 20 Bolton Street, Newcastle.

Dr. A. T. Roberts, over 5 Union Street, Newcastle.

Dr. Ethel Byrne, Royal Newcastle Hospital.

University Intelligence.

THE UNIVERSITY OF QUEENSLAND.

A NOTIFICATION has been published in the *Queensland Government Gazette* of November 26, 1949, that certain alterations have been made to the Statutes relating to the Board of Studies in Physiotherapy. These have been made necessary by the establishment of a degree of Bachelor of Applied Science in Physiotherapy.

Chapter XIXE of the Statutes is amended to read as follows.

BOARD OF STUDIES IN PHYSIOTHERAPY.

1. There shall be a Board to be called the Board of Studies in Physiotherapy, referred to in this Chapter as "the Board".

2. The Board shall consist of the President of the Professorial Board, the Dean of the Faculty of Science, the Dean of the Faculty of Medicine, the Lecturer-in-Charge of the Department of Physiotherapy, the Heads of Departments concerned with the teaching, the Director of Physical Education, one representative each of the British Medical Association, the Royal Australasian College of Physicians, the Royal Australasian College of Surgeons, the Royal Australasian College of Obstetricians and Gynæcologists, and the Physiotherapy Association, such representatives being approved by the Senate, and such other persons as the Senate may from time to time appoint.

3. Subject to the supreme control of the Senate, the duties of the Board shall be:

(*a*) To consider the curriculum of existing or projected courses leading to degrees, diplomas, or other awards in Physiotherapy.

(*b*) To propose such regulations or amendments thereof concerning the courses aforesaid as it considers desirable.

(*c*) In connection with such courses to propose the action to be taken on all matters brought before it relating to the work of students, exemptions, and the application of regulations concerning such courses, and to advise on the appointment of special lecturers, demonstrators and examiners.

(*d*) To report to the Professorial Board through the Faculty of Science its proposals or advice concerning the matters referred to above.

(*e*) To report to the Faculty of Science the results of the examinations in the courses referred to above and to make recommendations concerning supplementary examinations.

(*f*) To deal in accordance with the terms of reference with any matters referred to it by the Senate or any other superior authority.

4. The Lecturer-in-Charge of the Department of Physiotherapy shall be *ex officio* chairman of the Board. In the absence of the chairman from any meeting, the members present shall elect one of their members to be chairman of the meeting.

5. Five members of the Board shall form a quorum.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Holt, Geoffrey Charles, M.B., B.S., 1947 (Univ. Sydney), 68 Allanan Street, Young, New South Wales.

Diary for the Month.

DEC. 12.—Victorian Branch, B.M.A.: Executive Meeting.
DEC. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 13.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 14.—Victorian Branch, B.M.A.: Council Meeting.
DEC. 16.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 4.—Western Australian Branch, B.M.A.: Council Meeting.
JAN. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 10.—New South Wales Branch, B.M.A.: Council Quarterly.
JAN. 12.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 14.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 25.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 28.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 1.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 2.—South Australian Branch, B.M.A.: Council Meeting.
FEB. 3.—Queensland Branch, B.M.A.: Branch Meeting.
FEB. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 10.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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